Exploring gender differences in the prevalence of childhood externalising disorders

By

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Submitted in partial fulfilment of the requirements for the degree of Doctor of Psychology (Clinical)

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July, 2017
I am the author of the thesis entitled

**Exploring gender differences in the prevalence of childhood externalising disorders**

submitted for the degree of Doctor of Psychology

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Acknowledgements

First and foremost, I would like to thank my primary supervisor, Dr Merrilyn Hooley, for her dedication, wisdom, guidance and support with this thesis. Her supervision, advice and feedback have been invaluable and her teachings and influence will undoubtedly follow me throughout my career.

I would also like to thank my associate supervisor, Professor Jane McGillivray, and the co-authors of the studies presented in this thesis. Their expertise and guidance have been invaluable for both the quality of these studies and for my own learning.

I must also thank my family and friends. Their support throughout both the doctoral program and my overall university education has allowed me to remain focused and achieve beyond what I believed I could. Thank you for your patience, understanding, love and support.

I would also like to my doctoral program cohort at Deakin University. Thank you for being a source of support, friendship and laughter. I couldn’t have asked for a better group of people to travel this journey with.
List of peer-reviewed publications and conference presentations


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Abstract

The prevalence of childhood externalising disorders (EDs; attention-deficit/hyperactivity disorder [ADHD], conduct disorder [CD], and oppositional defiant disorder [ODD]) is consistently reported to be higher in boys than girls. This gender imbalance in ED prevalence rates raises questions as to possible gender-biased factors that influence the occurrence of the disorders. For example, it might be that factors associated with the development of EDs (e.g., the socialisation of certain ED-related behaviours by mothers and fathers through specific parenting behaviours that are related to ED development), and/or the diagnosis of EDs, differ between boys and girls to influence prevalence rates. To date, there has been little empirical investigation into these possibilities. This thesis sought to explore these possibilities via two aims: First, it sought to summarise the male:female prevalence ratios of EDs during middle childhood in non-referred children in order to synthesise and demonstrate patterns in the gender imbalance across EDs. Second, it sought to investigate how (i) parenting, and (ii) ED diagnosis, may differ between boys and girls and thus influence the gender imbalance in prevalence.

The first aim was addressed via Studies 1 and 2. Study 1, an overview of systematic reviews of the prevalence of EDs, demonstrated the male:female prevalence ratios for ADHD and CD during middle childhood in non-referred children ranged between 2.2-3.6:1, and 2.4-3.0:1, respectively. It also demonstrated that no such meta-analysed ratio for ODD existed at the time of the search. Study 2 addressed this gap and provided a meta-analysed male:female ratio of ODD prevalence during middle childhood in non-referred children (1.59:1).
The second aim was addressed via Studies 3 and 4. Study 3 focused on ADHD, and explored the prospective, bi-directional relationships between mothers’ and fathers’ Angry, Warm, and Consistent Parenting and child ADHD symptoms and diagnosis over five waves of data from the Longitudinal Study of Australian Children (LSAC; children aged 4/5 years at Wave 1; children aged 12/13 at Wave 5), and tested if these relationships differed for sons and daughters. A cross-lagged panel model demonstrated that (i) higher child ADHD symptoms at Wave 1 led to a global increase in less-than-optimal parenting at Wave 2 (increases in Angry Parenting and decreases in Warm and Consistent Parenting), and (ii) child ADHD symptoms and Angry Parenting shared a prospective, bi-directional relationship (whereby increases in one predicted increases in the other over time) during earlier years of development. Latent growth curve models demonstrated that increases in Angry Parenting across time were significantly predicted by increases in child ADHD symptoms. A logistic regression demonstrated that both mothers’ and fathers’ Angry Parenting at Wave 1 significantly predicted an ADHD diagnosis in children at Wave 3. None of these relationships differed between daughters and sons. Despite no child gender differences being found in these relationships, sons did have significantly higher rates of (i) Angry Parenting, (ii) ADHD symptoms, and (iii) ADHD diagnosis, at all waves. As Angry Parenting and child ADHD symptoms shared a prospective, bi-directional relationship in the earlier waves, this suggests that boys, compared to girls, may have an increased risk of ADHD development based on receiving higher levels of Angry Parenting during this period. This finding might help to account for the male preponderance in ADHD prevalence.
Study 4 examined if boys might be more likely to receive a diagnosis of an ED compared to girls, thus influencing the gender imbalance in ED prevalence. Again focusing on ADHD, Study 4 explored the notion that child gender may have become part of an ADHD diagnostic heuristic, with the prototypical representation of a child with ADHD including ‘being male’, which may lead to an increased chance of boys receiving an ADHD diagnosis and/or a decreased chance of a girl receiving an ADHD diagnosis. Participants (psychologists, psychiatrists and paediatricians) received two case vignettes of opposite gender across two time points both describing ADHD symptoms that either did, or did not, meet DSM 5 diagnostic criteria for ADHD. Results demonstrated that although diagnostic decisions did not appear to differ between vignettes describing a boy or a girl (suggesting that gender might not influence diagnostic decisions), participants demonstrated a clear propensity to diagnose ADHD even in vignettes where full diagnostic criteria were not met, demonstrating a concerning rate of ‘false positive’ diagnoses of ADHD.

The findings of this thesis are discussed in the final chapter in terms of their theoretical and clinical applications. The strengths and limitations of this thesis are also discussed, as are directions for future research.
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“It's pretty difficult to find any single factor that’s more predictive for some of these disorders than gender”.

Thomas Insel, during his time as Director of the National Institute for Mental Health in the United States of America, discusses widespread gender differences in mental health disorders (Holden, 2005, pg. 1574).
Chapter 1: Overview and introduction to thesis

1.1 Introduction and overview

Externalising disorders (EDs; at times also referred to as disruptive behavioural disorders [DBDs]) are a group of psychological disorders that occur, and are diagnosed, most often during middle childhood (6- to 13-years of age; Kessler et al., 2005), and are among the most common reasons for child referrals to mental health services (Reyno & McGrath, 2006; Zisser & Eyberg, 2010). Broadly, the presentations of EDs involve externalizing behaviours and a child’s inability or failure to control his or her actions in line with the expectations of others/society across various settings. Specifically, three psychological disorders come under the umbrella of EDs: attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiant disorder (ODD). The presentations of these disorders highlight their externalizing and disruptive nature and include a pattern of inattention and/or hyperactive-impulsive behaviours for ADHD, the persistent and repeated violation of social rules and the rights of others for CD, and a pattern of mood problems, defiant behaviour, and/or vindictiveness characterised by constant disobedience or hostility, for ODD (American Psychiatric Association [APA], 2013). These disorders also share high rates of comorbidity with each other (Biederman et al. 1993; Lavigne, LeBailly, Hopkins, Gouze & Binns, 2009; Sprafkin, Gadow, Weiss, Schneider & Nolan, 2007), suggesting that similar biological, psychological and/or social risk factors may influence their occurrence.

There are some caveats to the notion that ADHD, CD and ODD are EDs with DSM 5 re-classifying ADHD as a neurodevelopmental disorder, suggesting that social-level factors related to the development of ADHD may play a lesser role
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than they do for other EDs. A further to caveat involves the predominately inattentive subtype of ADHD (ADHD-I) that can potentially present with mostly internalising features. Despite the reclassification of ADHD in the DSM to a neurodevelopmental disorder, the symptoms and presentation of ADHD (particularly subtypes H/I and C) remain externalising. ADHD-I does contain predominantly internalising features in its presentation, and where possible the potential internalising nature of ADHD-I is acknowledged throughout this thesis in terms of how it might differ from the more externalising presentations of ADHD (i.e., ADHD combined type; ADHD-C; and the predominately hyperactive/impulsive ADHD subtype; ADHD-HI).

Aside from similarities in their externalizing and disruptive presentations, EDs share similar comorbidities (Burke, Loeber & Birmaher, 2002; Reid, Gonzalez, Nordess, Trout & Epstein, 2004), psychosocial treatments, and rates of mental health service utilization (Burke et al.; Greene et al., 2001; Kessler et al., 2005; Merikangas et al., 2011). One further commonality that EDs share is a male preponderance in their prevalence rates, which is found in both non-referred (i.e., community) and referred (i.e., clinical) samples of these disorders. For example, meta-analysed male:female prevalence ratios for ADHD, CD and ODD in non-referred samples are estimated at 2.2-3.6:1 (Erskine et al., 2013; Polanczyk & Jensen 2008; Willcutt, 2012; Wittchen et al., 2011), 2.4-3.0:1 (Erskine et al.; Wittchen et al.), and 1.59:1 (Demmer, Hooley, Sheen, McGillivray & Lum, 2016), respectively. The ratios from referred samples are often even more pronounced than those from non-referred samples (Owens, Cardoos & Hinshaw, 2015); for example, the male:female prevalence ratio for ADHD in clinical samples is suggested to be as high as 9:1 (APA, 1994). This gender imbalance in
the prevalence rates of EDs raises questions as to what influences the occurrence of these disorders (Derks, Dolan, Hudziak, Neale & Boomsma, 2007). For example, it might be that factors associated with the development of EDs (e.g., less-than-optimal parenting practices), and/or the diagnosis of EDs, differ between boys and girls and thus influence prevalence rates. To date, there has been little investigation into these possibilities (Bruchmüller, Margraf & Schneider, 2011; Gaub & Carlson, 1997; Kann & Hanna, 2000; Reid, et al., 2000).

One potential reason why this area has not been a strong focus of empirical attention may be due to the high reliance on male samples in existing ED research. As significantly more boys present clinically for EDs, males have traditionally been more readily accessible for recruitment into research (Gershon, 2002; Kann & Hanna, 2000). Considering this, it is likely that what is known about the development and diagnosis of EDs might more accurately represent boys with these disorders than it does girls (Gershon). Although there has recently been an increased interest into understanding the outcomes of EDs in girls (Gard, Owens & Hinshaw, 2016; Guelzow, Loya & Hinshaw; 2017; Owens & Hinshaw, 2016), there remains little known about how factors related to the development and diagnosis of EDs might differentially impact girls and boys or how these potential differences might help to explain the gender imbalance in ED prevalence rates. This thesis explores these possibilities.

The causes of the gender imbalance in the prevalence of EDs can be explored from a bio-psycho-social perspective, and thus may be driven by sex and/or gender. Sex is considered a biological construct that captures the anatomical, genetic, physiological and hormonal variations of species, often related to being male or being female (Johnson & Repta, 2012). Gender, on the other hand, is a
socially constructed concept that encapsulates the different roles, responsibilities, limitations and experiences afforded to individuals based on their sex and is often related to masculinity and femininity (Johnson & Repta). Gender is both produced and influenced by institutions such as social systems, education, media, as well as religious and political forces (Johnson & Repta). These forces also prescribe the gender roles (social norms, rules and standards that dictate appropriate behaviours and interests, and afford specific responsibilities, opportunities and limitations for males and females; Johnson, Greaves & Repta, 2007; Mahalik et al., 2003) that are deemed appropriate to, and associated with, males/masculinity and females/femininity.

From a biological perspective, it might be that biological pathways related to ED development differ for girls and boys (Burke et al., 2002; Derks et al., 2007). These biological explanations, however, are likely part of an epigenetic account whereby biology interacts with socialisation (Eme, 2007), supporting the importance of investigating how psychological- and social-level factors might also differ between girls and boys to influence the male preponderance in prevalence rates. From a psychological-level perspective, gender differences in personality characteristics linked to EDs (e.g., inhibitory control, impulsivity) may also help to account for the gender imbalance in ED prevalence if these were to be more common in boys than girls. However, given that the psychological attributes of personality characteristics likely have a biological basis, and in themselves are inferred/measured by the extent of observable externalising behaviours, there is an inescapable tautology in these explanations that impacts investigations at this level. Given these points, this thesis seeks to examine potential social-level factors that may influence the gender imbalance in ED
prevalence. Investigating factors at the social level will hopefully highlight potentially modifiable targets for psychosocial interventions and be of theoretical and clinical use for the profession of psychology.

The current thesis has two aims. The first aim is to summarise the current state of knowledge regarding the male:female prevalence ratios of EDs (ADHD, CD and ODD) during middle childhood in non-referred children. By summarising the male:female prevalence ratios it is hoped to synthesize and demonstrate the patterns in the gender imbalance across EDs. The second aim of this thesis is to investigate how (i) parenting, and (ii) diagnosis, two social-level factors related to EDs, may differ between boys and girls and thus influence the gender imbalance in prevalence.

1.2 A focus on middle childhood

This thesis focuses on the period of middle childhood, as this is when gender differences in externalising behaviours are most prominent. Prior to middle childhood girls and boys tend to demonstrate similar levels of ED-related behaviours (Alink et al., 2006; Hay, 2007; Keenan & Shaw, 1997; Loeber & Hay, 1997), and in adolescence these differences tend to diminish (Boylan, Vaillancourt, Boyle & Szatmari, 2007; Zoccolillo, 1993). Thus, if social-level factors do impact the gender differences in ED prevalence, this effect will possibly be most prominent during middle childhood. Also, given the average age of onset for ADHD, CD and ODD are all during middle childhood (Kessler et al., 2005), examining this period of development should (i) provide the largest available sample for investigation and, (ii) be generalizable back to the largest age-based population of children with EDs. Although it is possible that influences may occur during early development and that the outcome of these influences are
not seen until middle childhood, if social-level factors do impact the gender differences in ED prevalence, this effect will possibly be most prominent, and easiest to detect, during middle childhood, providing further rationale for investigating middle childhood.

1.3 A focus on ADHD

The two empirical studies of this thesis (Studies 3 and 4) focus on ADHD as their ED of interest. ADHD was selected for investigation for several reasons. First, due to the size limitations of this thesis, the inclusion of ADHD, CD as well as ODD in the empirical studies would have been impractical and well beyond its size constraints. Second, per the meta-analysed reviews outlined in Study 1 of this thesis (e.g., Erskine et al., 2013), ADHD sees the greatest gender differences in prevalence of all three EDs, thus suggesting the gender differences in the factors relating to ED development and/or ED diagnosis may be the most pronounced and detectable for this disorder compared to CD and ODD. It is hoped that the methods used in Studies 3 and 4 to investigate ADHD might be used in the future to guide investigations into CD and ODD.

1.4 Overview of proceeding chapters

This thesis commences with the current introduction and overview chapter that orientates the reader to the argument and structure that follows. Chapter 2 provides a brief overview of the negative outcomes associated with EDs, highlighting gender differences where they are known. Chapter 3 (Study 1) is an overview of existing systematic reviews and meta-analyses on the male:female prevalence ratios of EDs during middle childhood in non-referred children. The search revealed meta-analysed prevalence ratios for ADHD and CD, however not
for ODD. Chapter 4 (Study 2) addressed this gap in the literature and provided a meta-analysed male:female prevalence ratio of ODD during middle childhood in non-referred children. Study 2 has been published in the Journal of Abnormal Child Psychology (see Appendix A).

Studies 1 and 2 specifically investigated non-referred samples during middle childhood. As mentioned, ED research has been criticised for an over reliance on clinically referred male samples (e.g., Gershon, 2002; Kann & Hanna, 2000). As females are less likely to be referred, or present to services for EDs (e.g., Ohan & Visser, 2009; Polanczyk & Jensen, 2008; Schneider & Eisenberg, 2006), they tend to be under-represented, and more difficult to recruit, in research involving clinically referred samples. This brings into question the generalisability of current understandings based on referred samples, to females. Therefore, it is important to understand non-referred/community-based samples of EDs to increase understanding of how these disorders are impacting both genders.

Further, although the clinical male:female prevalence ratios are important to note, they say little about the estimated prevalence of disorders within the community or about the individuals who may not currently be receiving clinical intervention. Clinical ratios instead reflect only the number of males and females who are referred, diagnosed and/or treated for EDs (Bruchmüller, Margraf & Schneider, 2012). In contrast, non-referred samples are a more reliable gauge of the true impact of a disorder within the population, and are more easily generalised back to the population of interest.

Chapter 5 provides an overview of the development and diagnosis of EDs from a bio-psycho-social framework. Chapter 6 presents that argument that parents might play a role in the gender imbalance in child ED prevalence.
Specifically, it contends that (i) the associations between certain less-than-optimal parenting practices and EDs might be significant for sons but not daughters, and/or (ii) sons may receive higher levels of less-than-optimal parenting than daughters. Both suggestions might help to explain the male preponderance in ED prevalence. Chapter 7 (Study 3) explores these possibilities in a longitudinal analysis by examining child gender differences in the prospective, bi-directional associations between various parenting domains of mothers and fathers and child ADHD. Study 3 has been published in the Journal of Abnormal Child Psychology (see Appendix B).

Chapter 8 contends that boys may have an increased chance of receiving an ED diagnosis compared to girls due to the possibility that the ‘typical prototype’ of a child with an ED might include ‘being male’. Therefore, clinicians might consider children’s gender when assessing for a diagnosis. Chapter 9 (Study 4) explores this idea using a repeated-measures design, investigating potential differences in the diagnostic decisions of clinicians when assessing girls and boys. Study 4 has been submitted to the Journal of Child and Adolescent Psychology, and is currently under review. Chapter 10 provides a general discussion of the findings of this thesis, describes the theoretical and clinical implications of the empirical work, identifies its strengths and limitations, and suggests directions for future research.
Chapter 2: Negative outcomes associated with externalising disorders

2.1 Overview

Understanding causes of the gender differences in the prevalence of externalising disorders (EDs) may help inform targets for interventions for these disorders, and thus lessen detrimental outcomes associated with EDs. EDs negatively impact various domains of children’s functioning; for example, children with externalizing problems can expect poorer academic outcomes (Hinshaw 1992; Loe & Feldman, 2007; Masten et al., 2005; Reid, Gonzalez, Nordness, Trout & Epstein, 2004), impaired family and peer relationships (Bagwell, Molina, Pelham & Hoza, 2001; Hoza et al., 2005), and significant comorbid disorders and difficulties throughout life (Biederman et al., 2006; Burke, Loeber, Lahey & Rathouz, 2005; Ingram, Hechtman & Morgenstern, 1999), compared to typically developing children. The current chapter provides a brief overview of research in each of these domains and, where available, highlights where these outcomes differ based on child gender, to demonstrate domains in which functioning may improve for children if factors associated with the development and diagnosis of EDs are understood and targeted for intervention.

2.2 Academic outcomes

The academic performance of children with EDs is consistently lower than that of typically-developing children (Hinshaw 1992; Reid et al., 2004), with increases in externalising symptoms negatively associated with academic achievement in cross-sectional (Patalay, Fink, Fonagy & Deighton, 2016) and longitudinal studies (e.g., Masten et al., 2005). School and classroom issues for
children with ADHD can also involve increased rates of detention and expulsion and lower rates of graduation and/or higher education (Loe & Feldman, 2007). These academic issues are likely to impact the current and future opportunities available for affected children, exacerbating and extending the detrimental outcomes of EDs.

2.3 Interpersonal and familial outcomes

The presence of an ED can significantly impact a child’s relationships with others (Meltzer, Gatward, Goodman & Ford, 2000). For example, research has shown children with attention-deficit/hyperactivity disorder (ADHD) are less preferred by their peers (Hodgens, Cole & Boldizar, 2000) and are less popular within friendship groups (Hoza, 2007) than non-ADHD children. These peer difficulties often continue into adolescence (Bagwell et al., 2001) where the combination of ADHD and peer rejection has been shown to predict delinquency and anxiety, higher levels of nicotine use, a greater level of general impairment (Mrug et al., 2012), and an increased risk of eating pathology in girls (Mikami & Hinshaw, 2006). There are various mechanisms by which these social difficulties may occur. For example, the core symptoms of inattention and hyperactivity in ADHD might lead to a diminished ability for children to acquire social skills via observational learning, and/or to attend to social cues (Hoza, 2007) that are important in social learning, or to implement these skills once acquired. Further, common features of ADHD such as noncompliance and interfering in the classroom (Abikoff et al., 2002), as well as not paying attention during activities (Mrug, Hoza, Pelham, Gnagy & Greiner, 2007), may be poorly tolerated by peers and thus increase the rejection of children with ADHD (McQuade & Hoza, 2008; Paulson, Buermeyer, Nelson-Gray, 2005). It therefore appears that a bi-directional
relationship exists here, with peer rejection shown to be both a predictor (Miller-Johnson, Coie, Maumary-Gremaud & Bierman, 2002) and outcome (McQuade & Hoza, 2008; Paulson et al., 2005) of ED-related problems, thereby contributing to the maintenance of the disordered behaviours.

ADHD can also result in disturbed family, marital, and parent-child functioning, reduced parenting self-efficacy, and increased levels of parental stress and psychopathology (Johnston & Mash, 2001), family disorganisation, and family conflict (Schroder & Kelly, 2008). These children also often view their family environment as significantly more rigid and disengaged than do their typically developing peers, and report lower satisfaction with their family functioning (Pillay, 1998). These issues further negatively affect a child’s psychosocial adjustment, their beliefs about their own abilities, and academic achievement (Jones & Prinz, 2005).

2.4 Comorbid and future psychopathology

Children with EDs are also at a higher risk of further psychological difficulties. There are significant levels of comorbidity both within, and beyond, EDs. For example, ADHD has been shown to predict the onset of oppositional defiant disorder (ODD), while ODD often predicts the onset of conduct disorder (CD), anxiety and depression (Burke et al., 2005). Children with ADHD, when compared to non-ADHD children, demonstrate higher rates on composite diagnostic categories of anxiety disorders, antisocial disorders, developmental disorders, and substance dependence disorders (Biederman et al., 2006), and of attempted and completed suicide (Ljung, Chen, Lichtenstein & Larsson, 2014). It is also estimated that up to 60% of children with an ED retain some degree of
symptomology into adulthood (Ingram et al., 1999), suggesting negative outcomes can persist well past childhood.

2.5 Gender-specific negative outcomes

It has been demonstrated that although the outcomes of girls and boys with EDs may differ somewhat, both commonly experience negative outcomes and impairments (Gaub & Carlson, 1997; Gershon, 2002). For example, similar to boys, a prospective investigation following girls with ADHD found that regardless of whether ADHD remitted during childhood or persisted into adulthood, a childhood diagnosis of ADHD in girls predicts poorer academic outcomes, higher body mass index, and higher rates of unplanned pregnancy (Owens, Zalecki, Gillette & Hinshaw, 2017).

In a meta-analysis of gender differences in ADHD-related outcomes, Gaub and Carlson examined 17 studies (plus one dissertation being completed at the time) published before June 1994. Their findings demonstrated more similarities than differences in outcomes between genders. No significant gender differences were found in areas such as academic performance, impulsivity, social functioning, fine motor skills, or parental education or depression, however males were found to have higher levels of hyperactivity, and higher rates of internalising and externalising problems. Further, females with ADHD were found to have higher levels of intellectual impairment than their male counterparts. Gershon (2002) questioned the methodology of Gaub and Carlson’s review on the grounds of the limited number of included studies, and re-examined gender differences in ADHD by loosening inclusion criteria to include 38 studies for analysis. The main findings were largely similar between the two reviews, however Gershon noted
that females with ADHD tended to have a higher incidence of comorbidity with depressive and anxious symptoms than did males with ADHD.

In both reviews, it was concluded that the role of gender in ADHD, and particularly the impact of the disorder on girls, is still poorly understood, with Gaub and Carlson (1997) noting “The current literature leaves largely unanswered many of the most critical questions regarding the nature of ADHD in girls” (p. 1044). Based on these investigations it can be deduced that even if outcomes are not identical for girls and boys, girls with EDs still experience significant impacts and impairment, and investigations into the relationship between the development and diagnosis of EDs and child gender are needed to help identify potential targets for interventions that are relevant for both girls and boys.
Chapter 3: Study 1: Male:female prevalence ratios of externalising disorders: An overview of systematic reviews

3.1 Introduction and overview

There have been numerous systematic reviews and meta-analyses published that outline the male:female prevalence ratios of externalising disorders (EDs; e.g., Willcutt, 2012; Wittchen et al., 2011). The current chapter is an overview of systematic reviews that aims to outline the findings of these previously published reviews in a concise manner. Overviews of systematic reviews are a relatively new study design used to synthesize large areas of knowledge where multiple systematic reviews have already been undertaken and published (Silva, Grande, Martimbianco, Riera & Carvalho, 2012). It was expected that most existing systematic reviews would demonstrate higher prevalence rates for attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiant disorder (ODD) in boys than girls, supporting the notion that sex and/or gender is associated with factors related to the development and/or diagnosis of these disorders, and thus provide a rationale for empirical investigations of this thesis.

Given this, the aim of the study outlined in the current chapter was to systematically search for existing systematic reviews that presented meta-analysed male:female prevalence ratios of ADHD, CD and ODD during middle childhood in non-referred samples. It was hypothesized that literature that provided a male:female prevalence ratio would demonstrate a higher prevalence of EDs in boys compared to girls.
3.2 Method

3.2.1 Systematic search

The following databases were searched via Ebscohost: PsycInfo, Web of Knowledge, Medline Complete, Scopus, EMBASE, InfoRMIT, Psychological and Behavioural Sciences Collection, Cochrane Library, PubMed and ProQuest Health. A lateral search involving (i) a review of the reference lists of included reviews, and (ii) a by-hand search of Google Scholar, was also conducted. Box 1 outlines the terms used in the search. No restrictions were placed on publication dates. Reviews were limited to those written in English. Where possible within individual databases, appropriate limiters (e.g., ‘Childhood, 6-13’, ‘Review’) were used.

Box 1.

Search Terms

“Sex Difference” OR “Gender Difference” OR “Gender” OR “Male” OR “Female” OR “Boy” OR “Girl”

AND

“Rates of diagnosis” OR “Prevalence” OR “incidence” OR “Diagnosis rate” OR “Frequency”

AND

“Attention Deficit Hyperactivity Disorder” OR “ADHD” OR “Attention Deficit Disorder” OR “ADD” OR “Conduct Disorder” OR “CD” OR “Oppositional Defiant Disorder”

Studies were included if they met the following criteria:

(i) The review was a systematic review with a meta-analysis.

Systematic reviews without a meta-analysis were included if a
systematic review with a meta-analysis was located for that disorder. This was done to provide further understanding regarding the range and variability of the prevalence of disorders from non-meta-analytic systematic reviews.

(ii) The review was in relation to ADHD, CD and/or ODD

(iii) The studies included in the review contained non-referred samples (i.e., not clinical samples)

(iv) The age range of participants was within middle childhood (or had an average age between 6- to 13-years)

(v) The review reported a male:female prevalence ratio of the disorder, or prevalence rates for both boys and girls that could be converted to a ratio

Figure 1 presents a flow diagram of search results from initial search to final included studies. A total of nine reviews met the full inclusion criteria for this review (ADHD = 6; CD = 1; ADHD/CD combined = 2; ODD = 0).

3.2.2 Quality Assessment of Included Articles

Studies that met the full inclusion criteria were subjected to a quality assessment of their search procedure and methodology. The possible scores ranged from zero to eight, with higher scores representing better quality reviews. One point was assigned for each of the following criteria: the study (i) provided full numbers of papers at each search stage, (ii) outlined all information sources (e.g., names of databases searched, grey literature), (iii) outlined the search strategy so it was full replicable (e.g., search terms, limiters), (iv) outlined the inclusion and/or exclusion criteria, (v) had two authors involved at some stage of the process (e.g., study selection, data extraction), (vi) appropriately described its
methods of data extraction, (vii) conducted a quality analysis or assessment of bias for included studies, (viii) conducted a meta-analysis with risk of publication bias considered (e.g., funnel plot). Two researchers conducted these assessments separately, and disagreements were discussed until a consensus was reached. It was decided that all reviews would be included, despite some demonstrating poorer quality than others, in order to provide a more comprehensive understanding of the male:female prevalence rates of these disorders. However, the quality of each review should be considered when interpreting the results.
Figure 2. Flow diagram of search results

3.3 Results

3.3.1 Attention-Deficit/Hyperactivity Disorder

Table 1 presents the eight systematic reviews and their reported male:female prevalence ratios for ADHD. Five systematic reviews provided meta-analysed
male:female prevalence ratios (Erskine et al., 2013; Polanczyk et al., 2007; Polanczyk & Jensen, 2008; Willcutt, 2012; Wittchen et al., 2011), while three did not meta-analyse their included studies, instead reporting the male:female prevalence ratio ranges of the studies they included in their review (Catalá-López et al., 2012; Shooshtary et al., 2010; Skounti, Philalithis & Galanakis, 2007).

Five reviews provided data from multinational samples (Erskine et al., 2013; Polanczyk et al., 2007; Polanczyk & Jensen, 2008; Shooshtary et al., 2010; Willcutt, 2012), one from countries only within the European continent (Wittchen et al., 2011), and two provided estimates from single countries (Spain; Catalá-López et al., 2012; Iran; Shooshtary et al., 2010). In a review of Iranian studies, Shooshtary et al. estimated the male:female ratios to be 1:1.01 for the ADHD-Inattentive subtype and 1.3:1 for the ADHD-Hyperactive subtype, which suggests some variability in the male:female prevalence ratios across cultures when compared to European (3:1; Wittchen et al.) and multinational estimates (3.1:1; Erskine et al.). However, the estimates reported by Shooshtary et al. were derived from averaging the prevalence rates of their included studies rather than by more robust statistical methods, such as a meta-analysis. This impacts the validity of these estimates.

Two reviews included prevalence studies that used criteria from the diagnostic and statistical manual of mental disorders (DSM) III-R and/or DSM IV (Skounti et al., 2007; Willcutt, 2012), four reviews included studies that used criteria from various versions of both the DSM and international classification of diseases (ICD; Erskine et al., 2013; Polanczyk et al., 2007; Polanczyk & Jensen, 2008; Wittchen et al., 2011), one study included criteria from DSM-III-R, DSM-IV and other non-explicit sources (Catalá-López et al., 2012), and one study
included rates using Conners, Rutter, and CSI-4 questionnaires (Shooshtary et al., 2010). There did not appear to be notable trends or differences in the male:female prevalence ratios between reviews based on the measurements used in their included studies.
### Table 3.
**ADHD included Reviews and Descriptive Data**

<table>
<thead>
<tr>
<th>Review (Year)</th>
<th>Quality analysis score</th>
<th>Number of included samples</th>
<th>Age (years) range of included studies</th>
<th>Countries of included studies</th>
<th>Measures used in included studies</th>
<th>Male:female prevalence ratio</th>
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<tr>
<td>Catalá-López et al. (2012)</td>
<td>8</td>
<td>14</td>
<td>5-16</td>
<td>Spain only</td>
<td>DSM-III-R, DSM-IV or other/non-explicit</td>
<td>1:1.5 – 4:1</td>
</tr>
<tr>
<td>Erskine et al. (2013)</td>
<td>5</td>
<td>44 studies (75 prevalence estimates)</td>
<td>5-19</td>
<td>Multinational(a)</td>
<td>DSM-III onwards &amp; ICD-9 onwards</td>
<td>In 1990, 2005 and 2010: 3.1:1</td>
</tr>
<tr>
<td>Polanczyk &amp; Jensen (2008)</td>
<td>3</td>
<td>25</td>
<td>4-17</td>
<td>Multinational(b)</td>
<td>DSM-III, DSM-III-R, DSM-IV or ICD-9 onwards</td>
<td>2.4:1</td>
</tr>
<tr>
<td>Polanczyk et al. (2007)</td>
<td>8</td>
<td>44</td>
<td>&lt;18</td>
<td>Multinational(c)</td>
<td>DSM-III, DSM-III-R, DSM-IV or ICD-9 onwards</td>
<td>2.5:1</td>
</tr>
<tr>
<td>Shooshtary et al. (2010)</td>
<td>6</td>
<td>15</td>
<td>4-15 one included study contained a sample of 18-32</td>
<td>Iran only</td>
<td>Conners, Rutter, and CSI-4 questionnaires</td>
<td>Inattentive type: 1:1.01 Hyperactive type: 1.3:1</td>
</tr>
<tr>
<td>Study</td>
<td>Gender</td>
<td>Age</td>
<td>Prevalence</td>
<td>Region/Methodology</td>
<td>Diagnostic System</td>
<td>Ratio</td>
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<tr>
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<tr>
<td>Skounti et al. (2007)</td>
<td>0</td>
<td>20</td>
<td>2-20</td>
<td>Multinational^d</td>
<td>DSM-III-R &amp; DSM-IV</td>
<td>1:1 – 3:1</td>
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<tr>
<td>Willcutt (2012)</td>
<td>8</td>
<td>24</td>
<td>6-12</td>
<td>Multinational^e</td>
<td>DSM-IV</td>
<td>Overall: 2.3:1; Combined type: 3.6:1; Hyperactive type: 2.3:1; Inattentive type: 2.2:1</td>
</tr>
<tr>
<td>Wittchen et al. (2011)</td>
<td>4</td>
<td>12</td>
<td>6-17</td>
<td>All member states of the European Union (EU-27) plus Switzerland, Iceland and Norway</td>
<td>DSM-III, DSM-III-R, DSM-IV or ICD-10</td>
<td>3:1*</td>
</tr>
</tbody>
</table>

Notes:

^a Asia, Australasia, Caribbean, Europe, Latin America, North Africa, Middle East, North America, Oceania, Sub-Saharan Africa.

^b Finland, USA, Ethiopia, Germany, Brazil, Great Britain, Ukraine, Australia, The Netherlands, Sweden, England, South Africa, Venezuela, Colombia, Canada, Switzerland, Japan, Thailand, Scotland.

^c Africa, Middle East, Oceania, South America, Asia, North America, Europe, Worldwide.

^d Spain, Qatar, Thailand, Canada, USA, Turkey, New Zealand, England, Colombia, Brazil, Greece, Japan, Taiwan.

^e Nigeria, UK, South Arabia, Yemen, Spain, Iran, Brazil, Germany, Thailand, USA, Sweden, South Korea, Turkey, Australia, Malaysia, Congo, The Netherlands, China, Venezuela, Italy, Denmark, Greece, Gaza.

^"Best Estimate" provided by experts rather than meta-analysed ratio
3.3.2 Conduct Disorder

Table 2 presents the three systematic reviews and their reported male:female prevalence ratios for CD. Of these, one meta-analysed the findings of their included studies from multinational samples (Erskine et al., 2013), one provided a ‘best-estimate’ calculation based on expert opinion of studies published on European samples (Wittchen et al., 2011) and one reported on the range prevalence estimates in their included studies from multinational samples (Canino, Polanczyk, Bauermeister, Rhode & Frick, 2010). As was the case with ADHD, the reviews included studies with a range of assessment devices based on various diagnostic manuals, and ages tended to extend into adolescence.
Table 2
Conduct Disorder included reviews and descriptive data

<table>
<thead>
<tr>
<th>Review (Year)</th>
<th>Quality analysis score</th>
<th>Number of included prevalence estimates</th>
<th>Age range of sample in years</th>
<th>Countries of included studies</th>
<th>Measures used in included studies</th>
<th>Male:female ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canino et al. (2010)</td>
<td>6</td>
<td>23</td>
<td>4-18</td>
<td>Multinational&lt;sup&gt;a&lt;/sup&gt;</td>
<td>DSM-III-R and DSM-IV</td>
<td>1.1 – 7.5:1</td>
</tr>
<tr>
<td>Erskine et al. (2013)</td>
<td>5</td>
<td>56</td>
<td>5-19</td>
<td>Multinational&lt;sup&gt;b&lt;/sup&gt;</td>
<td>DSM-III to DSM-IV-R &amp; ICD-9 to ICD-10</td>
<td>2.4:1</td>
</tr>
<tr>
<td>Wittchen et al. (2011)</td>
<td>4</td>
<td>6</td>
<td>5-17</td>
<td>All member states of the European Union (EU-27) plus Switzerland, Iceland and Norway</td>
<td>DSM-III, DSM-III-R, DSM-IV and ICD-10</td>
<td>3:1*</td>
</tr>
</tbody>
</table>

<sup>a</sup>Spain, USA, Ethiopia, The Netherlands, China, Canada, Japan, Scotland, Great Britain.

<sup>b</sup>Asia, Australasia, Caribbean, Europe, Latin America, North Africa, Middle East, North America, Oceania, Sub-Saharan Africa.

<sup>*</sup>'Best Estimate' provided by experts rather than meta-analysed ratio.
3.3.3 Oppositional Defiant Disorder

The results of the systematic search demonstrated that no ODD reviews met full inclusion criteria. Two systematic reviews were found that presented sex differences in the prevalence of ODD (Boylan, Vaillancourt, Boyle & Szatmari, 2007; Canino et al., 2010), however, these studies reported only on trends and did not meta-analyse their data. As such, these were not included in the results as no published meta-analysis existed for ODD (inclusion criteria one). Thus, a summarised understanding (e.g., a meta-analysis) of the current state of knowledge regarding the male:female prevalence ratio of ODD is required.

3.4 Conclusion

The aim of the current chapter was to systematically search for existing systematic reviews that presented meta-analysed male:female prevalence ratios of ADHD, CD and ODD during middle childhood in non-referred samples to provide an overview of systematic reviews. It was hypothesized that literature that provided a male:female prevalence ratio would demonstrate a higher prevalence of EDs in boys than girls. This hypothesis was supported for ADHD and CD, with all meta-analysed ratios demonstrating a male preponderance in the prevalence of these disorders. This hypothesis was unable to be tested for ODD as no meta-analysed male:female prevalence ratio was found. This represents a significant gap in current research.

The finding of this overview supports the contention that sex and/or gender may be a significant risk factor for the development and/or diagnosis of ADHD and CD, and provides a rationale for the investigation into how
development and/or diagnosis factors related to these disorders might differ between boys and girls. Whether or not the male preponderance also occurs in the prevalence of ODD will not be well understood until a systematic review and meta-analysis is conducted for ODD prevalence studies to generate a single summary of the male:female prevalence ratio for this disorder. The proceeding chapter (Study 2) addresses this gap in the literature.
Chapter 4. Study 2: Sex differences in the prevalence of oppositional

defiant disorder during middle childhood: A meta-analysis

David H. Demmer\textsuperscript{1}, Merrilyn Hooley\textsuperscript{1}, Jade Sheen\textsuperscript{1}, Jane A. McGillivray\textsuperscript{1}

and Jarrad A. G. Lum\textsuperscript{1}

\textsuperscript{1}School of Psychology, Deakin University, Burwood, Australia

Published in: The Journal of Abnormal Child Psychology

Reference:


The \textit{online supplementary material} mentioned in Study 3 been included in Appendix C of this thesis.
Exploring gender differences in ED prevalence

Authorship Statement

**AUTHORSHIP STATEMENT**

1. Details of publication and executive author

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<th>Publication details</th>
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<td>Sex differences in the prevalence of oppositional defiant disorder during middle childhood: A meta-analysis</td>
<td>Journal of Abnormal Child Psychology</td>
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<th>School/Institute/Division if based at Deakin, Organisation and address if non-Deakin</th>
<th>Email or phone</th>
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</thead>
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<td>School of Psychology</td>
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</tr>
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2. Inclusion of publication in a thesis

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<th>If Yes, please complete Section 3 If No, go straight to Section 4.</th>
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3. HDR thesis author’s declaration

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<th>School/Institute/Division if based at Deakin</th>
<th>Thesis title</th>
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<td>David Hilton Demmer</td>
<td>School of Psychology</td>
<td>Exploring causes of gender differences in the prevalence of childhood externalising disorders</td>
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If there are multiple authors, give a full description of HDR thesis author’s contribution to the publication (for example, how much did you contribute to the conception of the project, the design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)

Conceptual development of paper, planning and completion of systematic search, evaluation of articles for inclusion, data extraction, assisted data analysis, drafting of manuscript, revisions, principal author.

I declare that the above is an accurate description of my contribution to this paper, and the contributions of other authors are as described below.

<table>
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4. Description of all author contributions

<table>
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<th>Name and affiliation of author</th>
<th>Contribution(s) (for example, conception of the project, design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)</th>
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<td>David Hilton Demmer</td>
<td>Conceptual development of paper, planning and completion of systematic search, evaluation of articles for inclusion, data extraction, assisted data analysis, drafting of manuscript, revisions, principal author.</td>
</tr>
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| Dr Merrilyn Hooley            | Thesis supervisor, conceptual development of paper, evaluated articles for inclusion, reviewed and feedback for manuscript. |
| Dr Jade Sheen                | Reviewing and feedback for manuscript. |
| Prof Jane McGillivray        | Reviewing and feedback for manuscript. |
| Dr Jarrod Lun                | Lead statistician, reviewing and feedback for manuscript. |
5. Author Declarations
I agree to be named as one of the authors of this work, and confirm:

i. that I have met the authorship criteria set out in the Deakin University Research Conduct Policy,
ii. that there are no other authors according to these criteria,
iii. that the description in Section 4 of my contribution(s) to this publication is accurate,
iv. that the data on which these findings are based are stored as set out in Section 7 below.

If this work is to form part of an HDR thesis as described in Sections 2 and 3, I further
v. consent to the incorporation of the publication into the candidate's HDR thesis submitted to Deakin University and, if the higher degree is awarded, the subsequent publication of the thesis by the university (subject to relevant Copyright provisions).

<table>
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<th>Name of author</th>
<th>Signature*</th>
<th>Date</th>
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<tr>
<td>David Hilton Demmow</td>
<td></td>
<td>14/3/2017</td>
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<tr>
<td>Dr Marilyn Hooley</td>
<td></td>
<td>14/3/17</td>
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<tr>
<td>Dr Jade Sheen</td>
<td></td>
<td>14/3/17</td>
</tr>
<tr>
<td>Prof Jane McGillivray</td>
<td></td>
<td>14/3/17</td>
</tr>
<tr>
<td>Dr Jarad Law</td>
<td></td>
<td>14/3/17</td>
</tr>
</tbody>
</table>

6. Other contributor declarations
I agree to be named as a non-author contributor to this work.

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<thead>
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<th>Name and affiliation of contributor</th>
<th>Contribution</th>
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</table>

* If an author or contributor is unavailable or otherwise unable to sign the statement of authorship, the Head of Academic Unit may sign on their behalf, noting the reason for their unavailability, provided there is no evidence to suggest that the person would object to being named as author.

7. Data storage
The original data for this project are stored in the following locations. (The locations must be within an appropriate institutional setting. If the executive author is a Deakin staff member and data are stored outside Deakin University, permission for this must be given by the Head of Academic Unit within which the executive author is based.)
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<th>Storage Location</th>
<th>Date lodged</th>
<th>Name of custodian if other than the executive author</th>
</tr>
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<tbody>
<tr>
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</tbody>
</table>

This form must be retained by the executive author, within the school or institute in which they are based. If the publication is to be included as part of an HDR thesis, a copy of this form must be included in the thesis with the publication.
Abstract

This review provides a meta-analysed male:female prevalence ratio of oppositional defiant disorder (ODD) during middle childhood in non-referred children. It also analyses sex differences in prevalence across cultures and over time. A systematic search for studies via the following sources was conducted: PsycInfo, Web of Knowledge, Medline Complete, Scopus, EMBASE, InfoRMIT, Psychological and Behavioural Sciences Collection, Cochrane Library, PubMed and ProQuest Health. The studies presented in two previous systematic reviews were also added to the search results. Inclusion/exclusion criteria were then applied and final studies were appraised for their methodological quality. Nineteen independent effect sizes met full inclusion criteria (aggregated sample \( N=44,107 \)). Overall, the prevalence of ODD was significantly higher in boys than girls (RR = 1.59, 95% CI [1.36, 1.86], \( p<.001 \)), with the male:female prevalence ratio found to be 1.59:1. Sex differences in prevalence were significant in Western (RR = 1.80, 95% CI [1.55, 2.10], \( p<.001 \)) but not non-Western cultures (RR = 1.08, 95% CI [-0.76, 1.53], \( p>.05 \)). Sex differences in prevalence were significant in studies published prior to and post the year 2000 (RR = 1.57, 95% CI [1.22, 2.02], \( p<.001 \); RR = 1.64, 95% CI [1.35, 2.00], \( p<.001 \)), and were consistent between these two periods (Q, 1 = 0.36, \( p=>.05 \)). The sex differences in ODD prevalence are discussed within the context of (i) predominant theories of sex differences in externalising behaviours, and (ii) departure from the sex-differences pattern found for other disruptive behavioural disorders.
4.1 Introduction

Oppositional Defiant Disorder (ODD) is characterised by a persistent pattern of angry/irritable mood, disobedient and hostile behaviour towards authority figures, and/or vindictiveness (American Psychiatric Association [APA], 2013), with population prevalence estimates ranging from 1.4% (Ashenafi, Kebede, Desta & Alem, 2001) to 12.3% (Cohen et al., 1993). Symptoms generally begin during the preschool years, with diagnosis typically occurring during middle childhood (i.e., 6- to 13-years-of-age; Kessler et al., 2005). ODD is associated with a range of detrimental outcomes including emotional and peer problems (Munkvold, Lundervold & Manger, 2011), family and social dysfunction (Greene et al., 2002), psychiatric comorbidity (Loeber, Burke, Lahey, Winters & Zera, 2000), high mental health service utilization (Cohen, Kasen, Brook & Struening, 1991), and is seen as a predictor in the development of conduct disorder (CD) and some personality disorders (Burke, Loeber & Birmaher, 2002; Holmes, Slaughter & Kashani, 2001; Loeber, Green, Keenan & Lahey, 1995; Maughan, Rowe, Messer, Goodman & Meltzer, 2004).

Due to commonalities between ODD, Attention-Deficit/Hyperactivity Disorder (ADHD) and CD in terms of symptoms, age of onset, comorbidities, psychosocial treatment, and mental health service utilization (Biederman et al., 2008; Burke et al., 2002; Greene et al., 2002; Kessler et al., 2005; Merikangas et al., 2011; Reid, Gonzalez, Nordess, Trout & Epstein, 2004), the three disorders are commonly grouped under the umbrella term of disruptive behavioural disorders (DBDs). A further similarity between DBDs is the marked sex differences in their prevalence rates, with all three disorders
more common in boys than girls. According to recent meta-analyses, the male:female prevalence ratio for ADHD is between 2.2:1 and 3.1:1 (Erskine et al., 2013; Willcutt, 2012; Wittchen et al., 2011), and between 2.4:1 and 3:1 for CD (Erskine et al., 2013; Wittchen et al., 2011) during middle childhood and adolescence. Although it is suggested ODD follows similar trends (Boylan, Vaillancourt, Boyle & Szatmari, 2007; Canino, Polanczyk, Bauermeister, Rohde & Frick, 2010), a meta-analysis to quantify this is yet to be conducted. This is an important investigation for several reasons. First, much of the published literature reports no statistically significant sex difference in ODD prevalence (e.g., Angold et al., 2002; Cohen et al., 1993; Leung et al., 2008; López-Villalobos et al., 2014; Mishra, Garg & Desai, 2014; Niemczyk, Equit, Braun-Bither, Klein & von Gontard, 2014; Park et al., 2014; Simonoff et al., 1997), thus clarity on whether one exists is needed. Second, confirming the male:female prevalence ratio of ODD invites speculation about, and offers potential avenues for, research into the aetiology and maintenance of the disorder (Aleman, Kahn, Selten, 2003; Zahn-Waxler, Shirtcliff & Marceau, 2008). The higher occurrence of ODD in males would imply that is something about being male that increases the risk of ODD developing/being maintained, and/or something about being female that protects against this risk.

4.1.2 Theories about sex differences

Various theories accounting for sex differences in the prevalence of ODD, other DBDs, and externalising problems in general, have been offered. These suggest biological factors (Caspi et al., 2002; Eme, 2007; Lahey et al., 2011), pathways in the development of DBDs (Crick & Zahn-Waxler, 2003;
Exploring gender differences in ED prevalence

Moffitt, 1993; Moffitt & Caspi, 2001), and differences in manifestations of externalizing problems (Crick & Zahn-Waxler, 2003) may be influential. Biologically, it is estimated that approximately half the variance associated with the development of externalising problems can be accounted for by biological factors (Derks, Dolan, Hudziak, Neale & Boomsma, 2007). For example, genetic research demonstrates the monoamine oxidase A (MAOA) and dopamine transporter (DATI) genes are linked to conduct behaviours (Caspi et al., 2002; Lahey et al., 2011), and that these may interact with environmental factors (such as parenting) to increases the difficulties with self-control that underlie delinquency behaviours (Watts & McNulty, 2014). Eme (2007) also discusses further potential leads in this area that warrant investigation; particularly why girls with congenital adrenal hyperplasia (CAH), a foetal disorder that leads girls to be exposed to male levels of androgens in utero, tend to have high rates of behavioural problems. Despite this, a common limitation of biological studies in this area is the failure to compare male and female participants, inadvertently hindering the exploration of a biological basis of sex differences in prevalence rates. Thus, how biological pathways in DBD development differ for boys and girls remains unclear (Burke et al., 2002; Derks et al., 2007; Moffitt et al., 1998).

Boys and girls follow different trajectories in the development of antisocial behaviours. Boys regularly follow either a child-onset pathway (the development of antisocial behaviours during early or middle childhood), or an adolescent-onset pathway (the development of antisocial behaviours during adolescence; Crick & Zahn-Waxler, 2003; Moffitt, 1993; Silverthorn & Frick, 1999). In contrast, the child-onset pathway in girls is rare (10:1 in
favour of boys; Eme, 2007; Moffitt & Caspi, 2001), with most girls who
develop antisocial behaviours doing so during adolescence (1.5:1 in favour of
boys; Moffitt & Caspi, 2001). This likely impacts the male:female prevalence
ratios of DBDs during early and middle childhood. It is suspected that girls
might be protected against child-onset due to various factors including their
erlier physical maturation, and better-developed language, social and
emotional skills compared to boys (Crick & Zahn-Waxler, 2003; Keenan &
Shaw, 1997). For both genders the child-onset pathway is particularly
important to understand as it is associated with greater risk of difficulties and
serious psychopathology into adulthood compared to the adolescent-onset
pathway (Moffitt & Caspi, 2001; Silverthorn & Frick, 1999).

Gender differences in DBD prevalence rates are also likely impacted by
boys and girls presenting with different manifestations of externalising issues
during childhood, despite a similar underlying mechanism driving both
presentations (Crick & Zahn-Waxler, 2003). This perspective is seen in the
idea that diagnostic criteria for ODD may fail to capture the complete range
of female presentations (Connor, 2002; Ohan & Johnston, 2005; Waschbusch
& King, 2006). For example, using sex-specific norms, Waschbusch and
King (2006) identified a group of untreated girls who did not meet DSM
criteria for an ODD diagnosis, yet had elevated ODD symptomology and
were almost as functionally impaired as girls who did meet criteria. Almost
no boys were identified when applying the same method. This suggests boys
are appropriately identified under existing DSM diagnostic criteria for ODD;
however there may exist a group of impaired girls who are missed by the
existing criteria, and thus may not be receiving the required assistance for
their difficulties. This finding is not surprising given diagnostic criteria are formulated from clinical samples, which for DBDs tend to be dominated by referred males (Gershon, 2002; Kann & Hanna, 2000).

Sex differences in the prevalence of DBDs may also be associated with sex differences in early risk factors. As an example, less-than-optimal parenting practices have been consistently associated with the development and maintenance of DBDs (Burke et al., 2002), including low levels of parental warmth and affection (Alizadeh, Applequist & Coolidge, 2007; Tripp, Schaughency, Langlands & Mouat, 2007), physical aggression (Stormshak et al., 2000), and high levels of hostility (Harold et al., 2013). Less-than-optimal parenting practices appear especially predictive of oppositional and aggressive behaviours in children (Stormshak, Bierman, McMahon & Lengua, 2000). As boys are more likely to receive less-than-optimal parenting than girls (de Ancos & Ascaso, 2011; Lloyd & Devine, 2006; Mahoney, Donnelly, Lewis & Maynard, 2000), boys appear to have higher exposure to this risk factor, possibly increasing the occurrence of ODD in boys. Although parenting practices appear to be a logical causal argument for the aetiology of DBDs, it is important to acknowledge the bi-directional relationship between parenting and child behaviour (Burke, Pardini & Loeber, 2008). Thus, higher rates of ODD in boys might be both causal, and consequential, of higher rates of less-than-optimal parenting.

4.1.3 Other influences on sex differences in prevalence rates

Extraneous influences that may contribute to sex differences in ODD prevalence are also an important consideration. Two of these are explored in the current meta-analysis: Culture and Time. Culture plays an important role
in the presentation and understanding of mental health disorders (Canino, Lewis-Fernández & Bravo, 1997). In their review of CD and ODD prevalence across cultures, Canino et al. (2010) analysed the effect of culture on CD and ODD prevalence rates, however found only methodological differences, and not geographical location, were associated with differences in prevalence rates between studies. Unfortunately Canino and colleagues were unable to quantify the male:female prevalence rate across cultures due to the limited data available, so how the sex differences in prevalence rates may differ across cultures remains unclear. Individual studies, however, do suggest sex differences are not universal across cultures. For example, in Western samples where traditional sex roles and gender expectations are considered to be minimised (e.g., USA, Great Britain), ODD typically trends to a higher prevalence in boys than girls (Angold et al., 2002; Bird et al., 2006; Costello, Mustillo, Erkanli, Keeler & Angold, 2003; Ford, Goodman & Meltzer, 2003; Simonoff et al., 1997). However, when investigating a Chinese sample, Leung et al. (2008) found a trend for a higher prevalence of ODD in girls than boys (10.4% versus 6.9%, albeit this difference was not statistically significant). It may be that cultural differences in tolerance of behaviours symptomatic of ODD in boys, girls, or both, may be impacting male:female prevalence ratios. For example, the attenuation in male dominance in Leung and colleagues’ Chinese sample may relate to a greater respect for traditional gender roles in non-Western cultures (Arnold, Choe & Roy, 1998; UNICEF, 2006). With higher expectation of demure behaviour in females, even subclinical externalising behaviours might be perceived as aberrant in girls and be reported as such on diagnostic instruments.
Understanding if sex differences in ODD prevalence have changed over
time may suggest social/environmental influences are important in the
disorder’s aetiology, referral, and/or diagnosis, as these influences can change
over shorter periods than biological aetiologies. The overall rate of conduct
problems more than doubled during the 1970s, 80s, and 90s (Collishaw,
Maughan, Goodman & Pickles, 2004), with the increase in girls’ delinquency
suggested to underlie this increase, and may have attenuated the sex
difference over time (Loeber et al., 2000). These trends over time are yet to
be examined for ODD.

4.1.4 The current review

The primary aim of the current review is to systematically search the
published literature to provide a meta-analysed male:female prevalence ratio
of ODD during middle childhood in non-referred children. Based on the sex
differences in the prevalence of other DBDs, we hypothesize that the
prevalence of ODD will be significantly higher in boys than girls. Two
secondary aims are (i) to examine cultural differences by providing
male:female prevalence ratios for Western and non-Western cultures
separately, and (ii) to examine potential changes in sex differences in ODD
prevalence over time.

4.2 Method

4.2.1 Systematic search

A systematic search for studies via the following databases was
conducted: PsycInfo, Medline Complete, Psychological and Behavioural
Sciences Collection, PubMed (all via EbscoHost), Web of Knowledge,
Scopus, EMBASE, InfoRMIT, Cochrane Library and ProQuest Health. The
studies reported in Boylan et al. (2007) and Canino et al. (2010) were also included in the search results. Neither of these two previous reviews provided a meta-analysed male:female prevalence ratio of ODD. Boylan et al. reported only individual study sex differences, and Canino et al. did not include child sex in their analysis due to the low number of studies that provided appropriate prevalence-by-sex data for extraction. A review of the reference lists of studies included in the meta-analysis was also conducted, as was a by-hand search of Google Scholar. The following search terms were used: “rates of diagnosis” or “diagnosis rate” or “prevalence” or “incidence” or “frequency” and “oppositional defiant disorder”. No restrictions were placed on the date of publication. The authors did not conduct a deliberate search for unpublished literature. It is unlikely this has biased our results as (i) much of the published literature already reports no significant sex differences in ODD prevalence, thus it seems non-significant findings are not a common reason to be rejected for publication, and (ii) the primary aim of most literature reporting ODD prevalence-by-gender is to provide an overall prevalence rate (i.e., collapsed across gender) for ODD as well as various other disorders. Thus, other similar studies are unlikely to have been rejected for publication due to non-significant sex differences in ODD prevalence as this is generally reported only as a supplementary/additional analysis and finding.

4.2.2 Study inclusion criteria

Several considerations were taken into account when planning this review and study inclusion criteria. First, to develop an understanding of sex differences in disorders it is important to examine non-referred samples (i.e., general population samples; Bruchmüller, Margraf & Schneider, 2012;
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Rutter, Caspi, & Moffitt, 2003). Referred samples say little about the true prevalence of a disorder, only reflecting the number of individuals who are diagnosed and/or treated, which may in fact be a function of other factors such as disorder severity, chronicity, and/or patterns of comorbidity (Rutter et al., 2003).

Second, as mentioned, the average age of onset for ODD is during middle childhood, and as suggested by others (e.g., Boylan et al., 2007), middle childhood sees the most pronounced sex differences in the prevalence of ODD before sex differences tend to even out during adolescence. Thus, the risk and/or protective factors impacting the occurrence of ODD in boys and girls may be most predominant during this period. In light of these points, investigating middle childhood specifically should allow for the most comprehensive examination and provide the most widely generalizable and useful findings, particularly in highlighting the unequal gender distribution of ODD and its possible gender-based risk and protective factors.

Studies were included in the final analysis if they met the following criteria. First, studies needed to contain a non-referred sample (i.e., not a clinical sample) obtained via probability sampling or total sampling. Second, the sample age range needed to be within middle childhood (i.e., six to thirteen years of age). In order to increase the number of included studies, to facilitate a more robust meta-analysis, studies with an age-range outside of middle childhood were included if they reported an average age range within the middle childhood years (e.g., 5- to 15-years-of-age with an average age of 10-years). Third, ODD prevalence needed to be based on a standardised measure derived from DSM (III, III-R, IV, IV-R, 5) or ICD (9, 10) diagnostic
criteria. Fourth, studies needed to report ODD point prevalence (i.e., not lifetime prevalence). Fifth, studies needed to report a male:female prevalence ratio of ODD, or numerical data that could be converted to a ratio for the purpose of meta-analysis.

4.2.3 Data extraction and meta-analytic procedures

Data were extracted from each study to permit calculation of a Risk Ratio (RR) and its variance of an ODD diagnosis given the sex of the participant. Specifically, female prevalence rates were extracted and converted to a percentage of the total female sample (for example, if the total sample of girls was 100, and 10 of those girls were identified as having ODD, the prevalence of ODD for girls in the study was 10%). The same procedure was then completed for the male prevalence rates. Standard errors were then calculated for each prevalence rate using the following formula: \( \text{SE}_p = \sqrt{p(1-p)/n} \). The conversion of extracted data to a RR was undertaken using Comprehensive Meta-Analysis Software Version 2.0 (Borenstein, Hedges, Higgins & Rothstein, 2005).

The RR was computed so that values greater than 1.0 indicate risk of ODD diagnosis was higher in males compared to females. RR values for the included effect sizes were averaged using a random-effect model (Hedges & Vevea, 1998). A random effects model was used to average study effect sizes. Thus it is assumed that differences between study-level effect sizes reflect random error and systematic influences. Specifically, a random-effects model assumes differences in study level effect sizes arise from two sources of error or variance: sampling error (within-study error), and true heterogeneity (between-study error). Between-study error arises from the
influence of one or more variables that systematically increase or decrease an effect size (e.g., geographical location, time). The weighting of studies in a random-effects model takes into account both within- and between-study error variance then assigns each study a weight based on the inverse of that variance (Borenstein, Hedges & Rothstein, 2007). This ensures the studies with higher numbers of participants are not simply assigned a higher weight in the meta-analysis based on their larger sample size.

4.3 Results

4.3.1 Search results

A flow diagram of search results is provided in Figure 1. The search was conducted by the first author and identified 1034 articles, with 27 additional records identified via a lateral search. A total of 607 articles remained after removing duplicates. The first author screened articles by title and/or abstract with 514 papers excluded at this stage (mostly due to studies not actually investigating ODD prevalence). The first author then examined 93 full text articles, with 74 excluded at this stage. The second author examined articles queried regarding their appropriateness for inclusion before a final decision was made. The reasons for exclusion at the full text stage are summarised in Figure 1, and each individual reason for exclusion is provided in the online supplementary material. The most common reason for exclusion was inappropriate data for extraction (e.g., 38 articles did not provide separate ODD prevalence rates for boys and girls). The remaining nineteen studies were appraised for their methodological quality and scientific rigour using a scale based on the Newcastle-Ottawa Scale adapted for cross-sectional studies (Wells et al., 2000). The scale criteria appraised those components of
studies that may be prone to introducing bias, and have been used to assess bias in previous meta-analyses (e.g., Herzog et al., 2013). Appraisal was conducted based on five components: sample (sampling procedure and sample size), validity of measurement tool, assessment process, number of respondents, and quality of statistical analysis (see Table 1 for specific criteria and Table 2 for scores of included studies). Each study could receive a possible score between zero and eight, with higher scores representing higher methodological quality. The first and second authors appraised studies independently, with disagreements resolved by discussion. Only one study (Erşan, Doğan, Doğan & Sümer, 2004) was assessed as high risk of bias and excluded from the analysis; this was because the name of the measurement tool was not provided and therefore could not be assessed for validity.
Figure 3. Flow diagram of search results
<table>
<thead>
<tr>
<th>Criteria</th>
<th>Two Points</th>
<th>One Point</th>
<th>No Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria 1. Selection (representativeness of sample; sample size)</td>
<td>Truly representative of the average in the target population (all subjects or random sampling)</td>
<td>Somewhat representative of the average in the target population (non-random sampling); Sample size justified and satisfactory</td>
<td>Selected group of users or No description of the sampling strategy;</td>
</tr>
<tr>
<td>Criteria 2. Measurement</td>
<td>Validated measurement tool</td>
<td>Non-validated measurement tool, but the tool is available or described</td>
<td>Inadequate description of the measurement tool</td>
</tr>
<tr>
<td>Criteria 3. Assessment</td>
<td>Interview conducted by a diagnostician (e.g., psychiatrist, psychologist) or diagnostician involved in the assessment process (e.g., overseeing assessments)</td>
<td>Interview/assessment conducted by a trained assessor (i.e., trained specifically for this study)</td>
<td>Interview/assessment not conducted by a trained/qualified assessor or not properly described or no interview conducted (e.g., survey only) or information not clear</td>
</tr>
<tr>
<td>Criteria 4. Respondents</td>
<td>2 or more respondents (e.g., parent and teacher)</td>
<td></td>
<td>One respondent</td>
</tr>
<tr>
<td>Criteria 5. Statistical test</td>
<td>The statistical test used to analyse the data is clearly described and appropriate (e.g., confidence intervals, p value)</td>
<td>The statistical test is not appropriate or Not described or incomplete.</td>
<td></td>
</tr>
</tbody>
</table>
Exploring gender differences in ED prevalence

Table 2.
Assessment of bias of studies meeting full selection criteria

<table>
<thead>
<tr>
<th>Study</th>
<th>Criteria 1</th>
<th>Criteria 2</th>
<th>Criteria 3</th>
<th>Criteria 4</th>
<th>Criteria 5</th>
<th>TOTAL SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Selection</td>
<td>Measurement</td>
<td>Assessment</td>
<td>Respondents</td>
<td>Test</td>
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<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>8</td>
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<tr>
<td>Angold et al. (2002)</td>
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<td>2</td>
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<td>0</td>
<td>1</td>
<td>6</td>
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<td>Ashenafi et al. (2001)</td>
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<td>2</td>
<td>0</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Bird et al. (2006)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>7</td>
</tr>
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<td>Carlson et al. (1997)</td>
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<td>0</td>
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<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
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<td>Costello et al. (2003)</td>
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<td>2</td>
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<td>1</td>
<td>1</td>
<td>7</td>
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<td>Erşan et al. (2004)**</td>
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<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
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<td>1</td>
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<td>1</td>
<td>0</td>
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<td>6</td>
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<tr>
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<td>1</td>
<td>1</td>
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<tr>
<td>López-Villalobos et al. (2014)</td>
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<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>8</td>
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<tr>
<td>Munkvold et al. (2011)</td>
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<td>2</td>
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<td>1</td>
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<tr>
<td>Niemczyk et al. (2014)</td>
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<td>0</td>
<td>1</td>
<td>6</td>
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<tr>
<td>Park et al. (2014)</td>
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<td>2</td>
<td>0</td>
<td>1</td>
<td>7</td>
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<td>Simonoff et al. (1997)</td>
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<td>1</td>
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<td>Sugawara et al. (1999)</td>
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<td>Zwirs et al. (2007)</td>
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<td>2</td>
<td>2</td>
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<td>1</td>
<td>7</td>
</tr>
</tbody>
</table>

**Excluded from analysis due issues with methodological quality and measurement of ODD**
4.3.2 Outline of included studies

Eighteen studies met full selection criteria and were assessed to be of sound methodological quality via the appraisal procedure, yielding 19 effect sizes for meta-analysis (Bird et al., 2006 contained two independent samples both with an independent effect size). Table 3 summarises the characteristics of each study. All studies were cross-sectional in design. The aggregated sample size was \( N = 44,107 \). Samples were from a broad range of countries, with the highest representation from the United States of America (USA; six studies). All samples had an average age range within middle childhood, however some included participants as young as five- (Ashenafi et al., 2001; Bird et al., 2006) and as old as 17-years-of-age (Angold et al., 2002). All measures used to determine the prevalence of ODD were based on DSM diagnostic criteria, and were responded to by parent and/or teacher and/or child via self-report measure and/or interview. The psychometric literature on these measures was examined by the first author in order to confirm that they were standardised, valid and reliable measures of child psychopathology.
<table>
<thead>
<tr>
<th>Study</th>
<th>Country/Region</th>
<th>N</th>
<th>Age range of sample in years</th>
<th>Measures</th>
<th>Prevalence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrés, Catala &amp; Gómez-Beneyto (1999)</td>
<td>Spain</td>
<td>387</td>
<td>10</td>
<td>KIDDY-SADS-E (Based on DSM-IV criteria)</td>
<td>4.9/2.5</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Parent only)</td>
<td></td>
</tr>
<tr>
<td>Angold et al. (2002)</td>
<td>USA</td>
<td>920</td>
<td>9 – 17</td>
<td>The Child and Adolescent Psychiatric Assessment (CAPA; based on DSM-IV criteria)</td>
<td>2.4/1.2</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>(Parent only)</td>
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<tr>
<td>Ashenafi, Kebede, Deste &amp; Alem (2001)</td>
<td>Ethiopia</td>
<td>1477</td>
<td>5 – 15</td>
<td>DICA (Based on DSM-III criteria)</td>
<td>1.2/1.6</td>
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<td></td>
<td></td>
<td></td>
<td>(Parent only)</td>
<td></td>
</tr>
<tr>
<td>Bird et al. (2006; USA)</td>
<td>USA</td>
<td>1138</td>
<td>5 – 13</td>
<td>DISC-IV (Based on DSM-IV criteria)</td>
<td>6.6/2.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Parent &amp; Child)</td>
<td></td>
</tr>
<tr>
<td>Bird et al. (2006; PR)</td>
<td>Peurto Rico</td>
<td>1353</td>
<td>5 – 13</td>
<td>DISC-IV (Based on DSM-IV criteria)</td>
<td>7.0/3.2</td>
</tr>
</tbody>
</table>

Table 3. Characteristics of studies included in meta-analysis
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Sample Size</th>
<th>Age Range</th>
<th>Assessment Tool</th>
<th>Reference</th>
<th>Male/Female Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carlson, Tamm &amp; Gaub (1997)</td>
<td>USA</td>
<td>2984</td>
<td>6 to 9</td>
<td>DSM-IV rating scale for ADHD and ODD</td>
<td>(Parent &amp; Child)</td>
<td>4/2</td>
</tr>
<tr>
<td>Cohen et al. (1993)</td>
<td>USA</td>
<td>541</td>
<td>10 - 13</td>
<td>DISC-1 (based on DSM-III-R criteria)</td>
<td>(Teacher only)</td>
<td>14.2/10.4</td>
</tr>
<tr>
<td>Costello, Mustillo, Erkanli, Keeler &amp; Angold (2003)</td>
<td>USA</td>
<td>6674</td>
<td>9 - 16</td>
<td>CAPA</td>
<td>(Parent &amp; Child)</td>
<td>3.1/2.1</td>
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<td>Ford, Goodman &amp; Meltzer (2003)</td>
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<td>10,438</td>
<td>5 - 15</td>
<td>The Development and Well-being Assessment (DAWBA; based on DSM-IV)</td>
<td>(Parent, Teacher &amp; Child over 11 years of age)</td>
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<td>6 - 8</td>
<td>ADIK (translated version of DICA; based on DSM-III criteria)</td>
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<tr>
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<td>Country</td>
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<td>Age Range</td>
<td>Tool/Measure</td>
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<td>1049</td>
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<td>6 - 11</td>
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<td>7 - 9</td>
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</tr>
<tr>
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<td>Simonoff et al. (1997)</td>
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<td>2762</td>
<td>8 - 16</td>
<td>CAPA (Based on DSM-III-R criteria)</td>
<td>3.9/3</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Sample Size</td>
<td>Age Range</td>
<td>Assessment Tools</td>
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<tr>
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<td>114</td>
<td>7 - 9</td>
<td>Child Assessment Schedule (CAS; based on DSM-III-R criteria)</td>
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<td>The Netherlands</td>
<td>2041</td>
<td>6 - 10</td>
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</tbody>
</table>
4.3.3 Assessment of Publication Bias

Publication bias was assessed using a funnel plot in conjunction with Egger’s test of asymmetry (Egger, Smith, Schneider & Minder, 1997). Funnel plots show the relationship between study level effect sizes and its standard error. The standard error is used to assess the precision of the effect size. Studies with larger standard errors provide a less precise estimate of the population parameter. Funnel plots indicate publication bias when the distribution of effect sizes is asymmetrical distributed around the weighted average effect size. This outcome indicates differences arise presumably because only significant (or even non-significant) findings were identified. A funnel plot showing the effect sizes included in the meta-analysis is presented in Figure 2. Egger’s test did not reveal significant levels of asymmetry in the effect sizes (Intercept = -0.91, t (17) = 1.00, p = .33).

Figure 4. Funnel plot outlining publication bias
4.3.4 Meta-analyses

A forest plot showing all 19 effect sizes and the weighted average is presented in Figure 3. The average weighted RR was found to be 1.59 and statistically significant (RR = 1.59, 95% CI [1.36, 1.86], *p*<.001), indicating an averaged male:female prevalence ratio of 1.59:1. As recommended by Higgins, Thompson, Deeks and Altman (2003), heterogeneity between effect sizes was assessed using the *I*² statistic. A moderate level of heterogeneity was found (*I*²=55.871). This suggests that 55.9% of differences between effect sizes represent true differences between those effect sizes and cannot be explained by chance.

As the male:female prevalence ratio was lower than expected based on those found for other DBDs, it was decided to remove the two effect sizes that demonstrated trends in the opposite-to-expected direction (i.e., a higher prevalence in females than males; Ashenafi et al., 2001 & Leung et al., 2008) to explore how these outlier effect sizes may have attenuated the overall ratio. The subsequent meta-analysis found the male:female prevalence ratio rose to 1.75:1 and remained significant (RR = 1.75, 95% CI [1.52, 2.00], *p*<.001).

The fourteen effect sizes drawn from Western cultures (see Table 3) were analysed and found to have a statistically significant average weighted RR of 1.8 (RR = 1.8, 95% CI [1.55, 2.10], *p*<.001). There was no significant sex difference in prevalence in effect sizes drawn from non-Western cultures (RR = 1.08, 95% CI [0.76, 1.53], *p*>.05). The effect size for the sex difference in Western studies was found to be significantly larger than for non-Western

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1 The term ‘outlier’ mistakenly went to print. ‘Outlier’ should instead be referred to as ‘aberrant data point’
studies ($Q, 1 = 9.95, p = .002; \text{ see Figure 4}$), demonstrating that the sex differences in ODD prevalence are more pronounced in Western than non-Western cultures.
## Exploring gender differences in ED prevalence

<table>
<thead>
<tr>
<th>Study name</th>
<th>Risk Ratio</th>
<th>95% C. I.</th>
<th>p-value</th>
<th>Risk greater in:</th>
<th>Weight (%)</th>
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<td></td>
<td></td>
<td>Lower</td>
<td>Upper</td>
<td></td>
<td>Female</td>
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<td>1.37</td>
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<td>2.16</td>
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<td>0.31</td>
<td>5.62</td>
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<tr>
<td>Ashenafi et al. (2001)</td>
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<td>3.00</td>
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<tr>
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<td>3.61</td>
<td>.002</td>
<td></td>
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<td>2.28</td>
<td>1.29</td>
<td>4.03</td>
<td>.005</td>
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<td>1.44</td>
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<td>1.86</td>
<td>.004</td>
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<td>Leung et al. (2008)</td>
<td>0.66</td>
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<td>1.16</td>
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<td>0.92</td>
<td>2.18</td>
<td>.115</td>
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</tbody>
</table>

**Average RR** 1.59 1.36 1.86 <.001

**Figure 3.** Forest plot showing study level and average weighted effect size (RR) for the prevalence of ODD in boys and girls.
Significant sex differences were found in the prevalence of ODD for studies published prior to (RR = 1.57, 95% CI [1.22, 2.02], p < .001) and studies published post the year 2000 (RR = 1.64, 95% CI [1.35, 2.00], p < .001). No significant difference was found between these two periods (Q, 1 = 0.36, p > .05; see Figure 5).

Figure 4. Forest plot showing combined effect sizes for non-Western and Western studies

4.4 Discussion

The primary purpose of the current meta-analysis was to provide a quantified male:female prevalence ratio for ODD during middle childhood in non-referred children. A systematic search of the literature yielded 19 independent effect sizes. The weighted average Relative Risk demonstrated significantly more boys are effected by ODD than girls, with a male:female prevalence ratio of 1.59:1 (which rose to 1.75:1 with the exclusion of two outlier studies). Two secondary aims were to explore the impact of Culture
and Time on the male:female prevalence ratio. Significant sex differences were found in ODD prevalence in Western but not non-Western cultures, as well as in studies published prior to and post the year 2000, with sex differences in prevalence remaining consistent between these two time periods.

Overall, our results confirm ODD follows a similar pattern to other DBDs in its prevalence, in that boys are more commonly affected than girls, but only in Western cultures. However, the sex difference found in the current results does not appear to be as pronounced as those reported in meta-analytic reviews for ADHD and CD (cf. ratios of 2.2:1 to 3.1:1 and 2.4:1 to 3:1 for ADHD and CD respectively; Erskine et al., 2013; Willcutt, 2012; Wittchen et al., 2011). Further, our overall male:female prevalence ratio of 1.59:1 is not reflective of the ratio previously reported for the child-onset developmental pathway of antisocial behaviour (10:1; Moffitt & Caspi, 2001), and instead appears almost identical to the adolescent-onset pathway (1.5:1; Moffitt & Caspi, 2001). These discrepancies warrant discussion. One possible explanation relates to the diagnostic characteristics of DBDs. ODD diagnostic criteria encompass mood problems (e.g., loses temper, touchy or easily annoyed, angry or resentful), argumentativeness and defiance (e.g., argues with authority figures, defies or refuses to comply with requests or rules) and vindictiveness (APA, 2013). These domains may in fact reflect attitudinal- and/or mood-orientated symptoms, which are inherently different from the action- and/or activity-orientated symptoms of CD and ADHD (excluding the ADHD inattentive subtype [ADHD-I]; e.g., physical fighting, physical cruelty, sexual behaviours, destruction of property for CD, and
problems with activity such as fidgeting, difficulty remaining seated, and inappropriate running and climbing for ADHD). Previous research has shown boys are more likely to display these externally-directed behaviours than girls, while girls tend to exhibit internally-directed symptomology, such as mood lability, shyness, withdrawal and hypersensitivity more so than boys (Carlson, Tamm & Gaub, 1997; Kann & Hanna, 2000; Lahey et al., 2000; Tiet, Wasserman, Loeber, McReynolds & Miller, 2001). Given the predominant presence of internalising-type symptomology in ODD, it might not be surprising that girls may be more prone to experiencing ODD than other DBDs, and/or boys less so. Examination of sex differences in ADHD-I adds support to this hypothesis. ADHD-I includes features such as inattention, distractibility and difficulties with concentration and memory (APA, 2013), which, similar to ODD, are less action- and activity-orientated than other ADHD subtypes and CD. Paired with this, the male:female prevalence ratio of ADHD-I is reported as lower than for ADHD-C (2.2:1 \textit{cf} 3.6:1 respectively; Willcutt, 2012), giving weight to the notion that action- and activity-orientated symptoms in DBDs may be less common in girls than boys.

Our finding that sex differences in ODD prevalence were only significant in Western countries is particularly interesting given the overall prevalence of ODD is found not to vary across cultures (Canino et al., 2010). Thus, the lack of sex differences in non-Western cultures, when compared to the significant sex differences found in Western cultures, may be due to either (i) a decreased prevalence of ODD in boys from non-Western cultures, or (ii) an increased prevalence of ODD in girls from non-Western cultures. If the latter
of these two is the case, this supports the hypothesis presented earlier that cultural differences in sex roles may be influential. Non-Western cultures where sex roles are more traditional may have a lower threshold for ODD behaviours in girls. Thus, subclinical ODD behaviours demonstrated by girls in Western cultures might be viewed as pathological in Non-Western cultures if they violate culturally derived sex-role stereotypes. This may lead to higher rates of referral and diagnosis of girls in non-Western cultures compared to Western cultures. Conversely, Western cultures may have increased tolerance for disruptive behaviours in girls (perhaps due to a higher tolerance of violations of gender norms) resulting in a lower risk of referral and diagnosis. Both of these interpretations would impact on prevalence rates in girls. Further investigations into why sex differences occur in Western but not non-Western cultures, despite overall prevalence rates not varying between the two (Canino et al., 2010), are needed.

Our results that a significant sex difference occurs in ODD prevalence, albeit only in Western cultures, invites speculation about why this difference exists and about the risk and/or protective factors associated with ODD. For example, cultural differences in prevalence-by-gender rates might suggest potentially modifiable socio-cultural factors that influence the aetiology, referral, and/or diagnosis of ODD in boys and/or girls. Further, the notion of sex-specific ODD diagnostic criteria to ensure appropriate diagnosis of both genders is worthy of exploration, given the hypothesis that ODD may manifest differently in boys and girls (Crick & Zahn-Waxler, 2003; Waschbusch & King, 2006). The possibility of an under diagnosis of girls, due to presentations that might not match current diagnostic criteria as
demonstrated by Waschbusch and King (2006), may help to account for girls’ lower ODD prevalence. Factors hypothesised to increase boys’ risk of DBDs also need further study. In particular, boys’ greater exposure to less-than-optimal parenting practices is an important avenue as (i) parenting practices are particularly predictive of oppositional and aggressive behaviours in children (Stormshak et al., 2000) and (ii) parenting practices represent a potentially modifiable influence.

We failed to find evidence of changes in sex differences in ODD prevalence in investigations published prior and post the year 2000. Across both time periods prevalence rates were higher in boys. It is possible this was due to the year chosen to split groups, however inspection of Figure 3, in which effect sizes are plotted chronologically, does not suggest an alternative split point, or evidence of time-related patterns. This finding does not fit with Loeber et al. (2000) who suggested increases in girls’ delinquency over time might have attenuated sex differences in externalising behaviours. Instead, this increase may have a greater impact on sex differences in CD prevalence given delinquent behaviours are more symptomatic of CD than ODD. The finding that sex-differences in ODD are consistent across time in the face of cultural and societal changes lends weight to biological accounts for the aetiology of ODD. Thus, future research into the biological aetiology of ODD and other externalising disorders should ensure they consider sex as a variable of interest in their investigations. Environmental factors (such as attitudes on gender) cannot be ruled out as a potential influence, as these have also remained somewhat stable in recent decades (Bolzendahl & Myers, 2004; Sayer, 2005; Twenge, 1997).
4.4.2 Limitations

The authors acknowledge several limitations of the current study. Despite our comprehensive search strategy, it is possible that literature was missed for various reasons (e.g., uncommon search/key terms). There was also little consistency between included studies in their measures used and the number of respondents. This may have led to differences in the ODD construct being measured, impacting the validity of our results. Various studies engaged individual, dyad or triad respondents. Although it might be assumed that more respondents would increase the accuracy of estimates, there is often little consistency in ratings between multiple respondents, affecting the accuracy of combined ratings (O’Neill, Schneiderman, Rajendran, Marks & Halperin, 2014; Stokes, Mellor, Yeow & Hapidzal, 2014). This issue may help to account for the moderate level of heterogeneity of our results.

Only eight of the 19 effect sizes included here reported significant sex differences in the prevalence of ODD. Given this inconsistency we attempted to identify methodological parameters that might distinguish the studies that found significant differences from those that did not find significant differences. Data summarised in Table 3 were examined for trends, including geographical location of the sample (including cultural considerations), age range of the participants, sample size, measures used, and number of respondents. No obvious trends or patterns were discernible.

4.4.3 Conclusion

The current review found an overall male dominance in ODD prevalence, with this result holding across Western, but not non-Western, cultures and across time. Attention should now be paid to investigating the possible
reasons for these sex differences in ODD prevalence from a bio-psycho-social perspective. Several hypotheses have been suggested here and warrant future investigation, particularly the notion of cultural differences in prevalence-by-gender rates, gender-biased diagnostic criteria, and the role of gender differences in early ODD risk factors. Given that ODD is a predictor of later psychopathology, such as CD and personality disorders (Burke et al., 2002; Holmes et al. 2001; Loeber et al., 1995; Maughan et al., 2004), ensuring that the male dominance is not due to under- or misdiagnosis of ODD in females’ early in development is critical to minimize their risk of lifelong detrimental effects.

David H. Demmer declares he has no conflict of interest. Merrilyn Hooley declares she has no conflict of interest. Jade Sheen declares she has no conflict of interest. Jane A. McGillivray declares she has no conflict of interest. Jarrad A. G. Lum declares he has no conflict of interest.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

This article does not contain any studies with human participants performed by any of the authors. For this type of study formal consent is not required.
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oppositional defiant disorder and conduct disorder in ADHD boys: Findings from a controlled 10-year prospective longitudinal follow-up study. *Psychological Medicine, 38*, 1027-1036.


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Exploring gender differences in ED prevalence


Exploring gender differences in ED prevalence


O’Neill, S., Schneiderman, R., Rajendran, K., Marks, D. & Halperin, J. (2014). Reliable ratings or reading tea leaves: Can parent, teacher, and
Exploring gender differences in ED prevalence


Chapter 5: Bio-Psycho-Social factors related to the development and diagnosis of externalising disorders

Chapters 3 and 4 (i.e., Studies 1 and 2) established that there is a male preponderance in the prevalence of attention deficit hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiant disorder (ODD) during middle childhood in non-referred samples. This chapter explores potential causes for this gender imbalance in externalising disorder (ED) prevalence from a bio-psycho-social perspective. It reviews current understandings regarding the development and diagnosis of EDs, and how these factors might differ between girls and boys to influence the male:female prevalence ratios. In general, an overview of each factor associated with EDs is discussed before an argument is made (where evidence exists) about how the factor might differ between girls and boys and thus influences the gender differences in ED prevalence.

5.2 Gender differences in the developmental pathways of externalising disorders

Theorists have proposed two distinct pathways to the onset of antisocial behaviours, such as those symptomatic of CD and ODD, and these are based on the developmental period at which symptoms first emerge. The childhood-onset pathway involves antisocial behaviours beginning early in development, generally well prior to adolescence, which commonly leads to antisocial behaviours becoming increasingly more severe over time (Crick & Zahn-Waxler, 2003; Moffitt, 1993). Alternatively, the adolescent-onset pathway involves a more sudden onset of antisocial behaviours around the adolescent years (Crick & Zahn-Waxler; Moffitt). The childhood-onset
pathway, at times referred to as life-course-persistent pathway given the earlier age of onset and continued presence of symptoms, is associated with more significant and ongoing difficulties throughout life compared to the adolescent-onset pathway, whereas the adolescent-onset pathway is associated with antisocial presentations that are relatively limited to adolescence (Moffitt & Caspi, 2001).

The two pathways introduced above do not appear to explain female trajectories of anti-social behaviour development as well as they explain male trajectories, as the childhood-onset pathway is rare in girls. Instead, girls generally do not manifest antisocial behaviours until the onset of puberty and adolescence, suggested to be a delayed-onset pathway (Silverthorn & Frick, 1999; Silverthorn, Frick & Reynolds, 2001). It is argued that a variety of bio-psycho-social factors protect girls from the childhood pathway (e.g., earlier social skills development compared to boys), while a variety of factors associated with the onset of puberty and adolescence (e.g., the onset of menstruation in girls, and the view that puberty is a negative experience for girls yet a positive experience for boys) lead to the display of antisocial behaviours in girls with pre-existing vulnerabilities (Keenan & Shaw, 1997; Silverthorn et al.).

5.3 Biological-level explanations

5.3.1 Genes and Neurotransmitters.

It is estimated that approximately half of the variance associated with the development of EDs can be accounted for by biological factors (Derks, Dolan, Hudziak, Neale & Boomsma, 2007). The role of genes and heritability in the development of EDs has been outlined in genetic, twin, and adoption
Exploring gender differences in ED prevalence

studies, and has been summarised in several reviews (e.g., Bobb, Castellanos, Addington & Rapoport, 2006; Farone & Doyle, 2000; Hicks, Krueger, Iacono, McGue & Patrick, 2004; McCracken, et al., 2000; Slutske, et al., 1997; Thapar, Cooper, Eyre & Langley, 2013; Thapar, Hervas & McGuffin, 1995). These reviews have focused mainly on ADHD, finding the DRD4, DAT1, DRD5, 5-HTT and HTR1B genes that affect the dopamine D4 and D5 receptors and the dopamine and serotonin transporters are important in the development of ADHD.

The biological influences for conduct and oppositional problems are less clear (Burke, Loeber & Birmaher, 2002), but serotonin has been identified as playing a role in violence- and aggression-based ED behaviours (Burke et al., 2002; Olvera, 2002). Further, genetic research demonstrates the monoamine oxidase A (MAOA) and dopamine transporter (DATI) genes are also linked to conduct behaviours (Caspi et al., 2002; Lahey et al., 2011), and that these may interact with environmental factors, such as parenting practices, to increase the difficulties with self-control that underlie delinquency behaviours (Watts & McNulty, 2014).

Eme (2007) has reviewed biological factors that may influence sex differences in CD and how these biological differences may interact with gender socialisation. For example, in comparison to females, males are generally taller (Pinker, 2002), stronger (Buss, 2004), have greater oxygen intake, more muscle, and larger organs (e.g., lungs and hearts) that are essential for physical activity (Holden, 2004). Eme hypothesised that over the course of human existence these physical differences have interacted with social needs to influence the roles held, and more effectively completed by,
males and females. In short, Emes argues that over time the male physique has been more biologically suited to roles involving ED-related behaviours (e.g., physical violence, hunting, war) than the female physique, leading men to engage in these roles more so than females.

### 5.3.2 Neuroanatomy

Neuroanatomical factors have been associated with EDs. For example, boys and girls with ADHD show disruptions in regions of the frontal lobe, the basal ganglia, the cerebellar hemispheres, and a sub-region of the cerebellar vermis, as well as a decreased volume of overall white matter (for a meta-analysis of volumetric studies in ADHD see Castellanos & Acosta, 2004; Krain & Castellanos, 2006). The association between these neuroanatomical factors and the behavioural, neuropsychological and phenotypic expressions of ADHD remain underexplored, and it remains unclear as to whether these brain differences are causal, or consequential, of ADHD. Research into brain areas associated with violence and impulsive aggression is often used to support a biological account of CD and ODD (Olvera, 2002), and again while causal influences are difficult to assert, there is some suggestion that interference with certain brain areas may increase CD- and ODD-related behaviours. For example, Burke et al. (2002) summarise evidence demonstrating that frontal lobe damage can play a role in the aggressive behaviours associated with EDs, while deficits in amygdala functioning are associated with disruptions in the interpretation of social cues, which may lead to antisocial responses.

Subtle sex differences in the brain function of children with EDs have been noted, demonstrating that this aspect of ED development may be
somewhat different between the sexes. However, this research again has tended to focus on ADHD. For example, boys with ADHD exhibit a less right-lateralized-frontal-activation pattern when compared to healthy controls, whereas girls exhibit a more right-lateralized-frontal-activation pattern (Baving, Laucht & Schmidt, 1999). Electroencephalography (EEG) studies have shown that girls with ADHD show elevated coherence in frontal and temporal areas, and theta wave enhancement localised to frontal areas of the brain, while boys with ADHD demonstrate a more diffuse theta wave enhancement (Barry, Clarke, McCarthy & Selikowitz, 2006; Hermens, Kohn, Clarke, Gordon & Williams, 2005). Research involving functional neuroimaging of adult brains has also demonstrated that males with ADHD may have a higher degree of impairment in the development of the cerebellar-prefrontal-striatal networks than females with ADHD (Valera et al., 2010), but it is not clear whether the finding can be generalized to ADHD-affected children. Taken together, these findings suggest a more extensive and severe neurodevelopmental phenotype in males with ADHD compared to females with ADHD (Davies, 2014).

Whether biological and neuroanatomical factors are a cause, or consequence, of EDs, or whether they co-occur with EDs as a result of the third factor is still not clear; however, some prospective evidence supports a causal relationship (Burke et al., 2002). Rutter (2001), however, suggests that biological and neuroanatomical factors alone cannot predict the development of disorders such as ADHD, and by extension, are unlikely to adequately account for the significant gender differences seen in the prevalence of EDs. Unfortunately, a common limitation of biological studies of the development
of EDs is the failure to compare male and female participants, inadvertently hindering the exploration of a biological basis of sex differences in EDs. For this reason, how biological pathways in ED development differ for boys and girls remains unclear (Burke et al., 2002; Derks et al., 2007; Moffitt et al., 1998).

5.4 Psychological-level explanations

From a personality perspective, high levels of impulsivity and negative emotionality (e.g., reactivity to stress, emotional lability), and low levels of behavioural inhibition and constraint (e.g., responsibility, dependability, moral consciousness), are related to the development and maintenance of externalising problems (Burke et al., 2002; Cukrowicz, Taylor, Scharschneider & Iacono, 2006; Sanson & Prior, 1999). However, given that the psychological attributes of impulsivity and inhibitory control are inferred from the extent of externalising behaviours, the relationships between ‘internal’ psychological characteristics and ‘external’ behaviours appear to be circular. For example, it might be inferred that a child is impulsive because he or she manifests reckless behaviour, so it is therefore inappropriate to suggest that impulsivity might be the cause of the reckless behaviour. It has been argued that some temperament and personality characteristics, such as activity level and the Big Five dimensions of personality, are inherently different from the symptoms of externalizing psychopathology, and therefore may be more appropriate to discuss in terms of causal processes (Muris & Ollendick, 2005). For example, males have higher levels of activity than females (Eaton & Yu, 1989; Riddoch et al., 2004; Van Mechelen, Twisk, Post, Snel & Kemper, 2000), and have a small but increased
likelihood to engage with novel situations and events (i.e., ‘approach’
tendencies) than females (Else-Quest, Hyde, Goldsmith & Van Hulle, 2006).
However, these examples can also be considered as dimensions of EDs (e.g.,
the problems with activity in ADHD), and therefore invoking cause-effect
relationships between these constructs would also involve circular reasoning.
Conscientiousness, neuroticism and agreeableness, three of the Big Five
Dimensions of personality are also related to EDs. For example, ADHD has
shown to be related to low conscientiousness and neuroticism, and both
ADHD and oppositional behaviours are shown to be associated with low
agreeableness (Nigg et al., 2002). It is possible, however, that a third factor
(e.g., neuroanatomical, neurochemical, environmental) underlies the outward
expressions captured by these personality attributes and symptoms of
psychopathology.

Gender differences in levels of empathy and guilt are also thought to be
involved in the development of EDs (Bybee, 1998; Grusec & Hastings, 2014;
Keenan, Loeber & Green, 1999). Compared to boys, girls tend to spend more
time ruminating over negative behaviours and report higher levels of guilt
related to their inconsiderate behaviours, such as those behaviours seen in CD
and ODD. This rumination may serve as a deterrent for disruptive and anti-
social behaviours and help girls engage in higher levels of behavioural self-
regulation than boys.

5.5 Social-level explanations

Although EDs have a high heritability component (e.g., ADHD being
two to eight times more likely in a first degree relative of an individual with
ADHD; Faraone et al., 2005), heritability estimates include not only genetic
Exploring gender differences in ED prevalence

influences, but considerations of shared environments and gene-environment interactions (Thapar et al. 2013) whereby the environment influences the occurrence and/or expression of certain genetically-determined presentations and behaviours. As such, social influences are suggested to be fundamentally important in the development of EDs (Meier, Slutske, Heath & Martin, 2011). While numerous social-level factors related to EDs can be examined, the empirical studies of this thesis investigate the development of EDs via parenting and the diagnosis of EDs. The arguments for how these social-level factors might be differentially associated with EDs based on child gender is introduced here before being explored thoroughly in Chapter 6 for parenting and Chapter 8 for the diagnosis of EDs.

5.5.1 The development of externalising disorders via less-than-optimal parenting

One factor that has been consistently associated with the development of EDs is less-than-optimal parenting (e.g., Keown, 2012; Lifford, Harold & Thapar, 2008). Different relationships have been found for parenting and symptoms of ADHD, CD and ODD, and therefore these different relationships are considered throughout this thesis. However, less is known about how this association might differ based on child gender. This thesis explores two ways in which the association between less-than-optimal parenting and EDs might differ based on child gender. First, it might be that this association is significant for boys but not girls. To date, this possibility has received minimal empirical attention and has mostly focused on CD, with inconsistent findings making it difficult to draw conclusions (e.g., Keenan et al., 2010; Tung, Li & Lee, 2012). As such, further investigation is needed to
clarify this association for conduct problems, and explore this same possibility for other EDs. Second, it might be that boys are exposed to higher levels of less-than-optimal parenting (that is associated with higher levels of ED symptoms) than girls. Both possibilities would lead to boys having an increased risk of ED development, and potentially influence the male preponderance in ED prevalence. These ideas are the focus of Study 3 and the rationale for this is more thoroughly explored in Chapter 6.

**5.5.2 The diagnosis of externalising disorders**

Issues regarding ED diagnosis might also impact the male preponderance in ED prevalence if boys are more likely than girls to receive an ED diagnosis. Children exhibiting more *observable or obvious* symptoms (i.e., those associated with EDs) might have an increased chance of being noticed by potential referral sources (e.g., parents, teachers) and therefore have an increased chance of referral and diagnosis. Gender differences in the presentation of EDs may contribute to this. Boys, when compared to girls, tend to present with more disruptive and overt manifestations of EDs (e.g., the hyperactive/impulsive behaviours of ADHD; Biederman et al., 2002), which may be more noticeable to caregivers and teachers than the more covert behaviours displayed by girls (e.g., the inattentive behaviours of ADHD; Quinn, 2005). Similar gender differences in symptoms are seen in the presentation of conduct problems (Kann & Hanna, 2000). For example, males with CD tend to engage in externally directed behaviours such as physical aggression, acts that are harmful to the environment and others, and destruction of property, while girls with CD tend to exhibit the disorder’s issues with deficient affect (e.g., shyness, withdrawal; Lahey et al., 2000;
Exploring gender differences in ED prevalence

Tiet, Wasserman, Loeber, McReynolds & Miller, 2001). Again, boys’ more disruptive and potentially more noticeable presentations of conduct disorder may also lead to increased chances for referral, and potentially diagnosis, compared to girls. It is also possible that there may be differing symptom thresholds and/or diagnostic criteria needed for diagnosing CD, in a manner similar to that proposed to occur in the diagnosis of ODD in girls and boys (e.g., Connor, 2002; Ohan & Johnston, 2005; Waschbusch & King, 2006). That is, the symptom threshold may be inappropriate for girls if the mean level of conduct problems is lower for girls than for boys.

Once referred, it might be that the chance of an ED diagnosis differs based on the gender of the child. The dramatic differences in the male:female prevalence ratios of EDs between referred and non-referred samples (e.g., 9:1 versus 3.6:1 for ADHD; APA, 2013; Willcutt, 2012) suggest such a gender bias in referral and/or diagnosis might be occurring. Specifically, they suggest that a substantial number of girls with ED presentations are not being referred for assistance and/or are not being given a ED diagnosis when potentially appropriate, and/or, a substantial number of boys without true ED presentations are being referred and diagnosed when potentially inappropriate. It has been suggested that this gender bias could be due to gender being considered by diagnosticians when they are assessing for a diagnosis; for example, the ‘prototypical representation’ of a disorder such as ADHD may include ‘being male’, increasing the chances of a boy receiving a diagnosis over a girl even when presentations are identical (Bruchmüller, Margraf & Schneider, 2012). This would thereby influence the male
preponderance in EDs for which this is occurring. This idea is the focus of Study 4 and the rationale for this is more thoroughly explored in Chapter 8.

5.6 Conclusion

Despite other potential social-level influences (e.g., teachers, peers), parenting and diagnosis were chosen for empirical investigation in this thesis for several reasons. First, there has been little investigation into how these factors might differ between girls and boys. Second, both parenting and diagnosis represent potentially modifiable factors. Therefore, investigating these factors could provide suggestions for psychosocial treatments of EDs and provide utility for the profession of psychology. Third, they examine two different levels of social factors, with parenting associated with the development of ED-related behaviours, and diagnosis associated with the labelling and official presence of the disorder. This allowed for a broader investigation into social-level factors.
Chapter 6: Parenting, externalising disorders, and child gender

As introduced in Chapter 5, parenting might contribute to the gender imbalance in externalizing disorder (ED) prevalence if (i) less-than-optimal parenting practices are related to the development of EDs in boys but not girls, and/or (ii) boys receive higher levels of less-than-optimal parenting (that are related to ED development) than girls. These suggestions are explored in this chapter.

6.2 Potential child gender differences in the associations between parenting practices and externalising disorders

There is considerable evidence that less-than-optimal parenting practices are associated with the development of externalising disorders. For example, higher ED symptoms are related to higher levels of controlling parenting behaviours (e.g., the application of pressure by parents through punishment, commands, or coercive interactions; Rogers, Wiener, Marton & Tannock, 2009), higher parental overprotection (e.g., discouragement of behavioural freedom and autonomy; Chang, Chiu, Wu, & Gau, 2013), lower levels of parental warmth and affection (e.g., displays of warmth and involvement; Gau, 2007; Tripp, Schaughency, Langland, & Mouat, 2007), inconsistent parenting (e.g., consistent reinforcement and punishment of specific behaviours; Cussen et al., 2012), lower parental responsiveness (e.g., responsiveness to child’s requests for attention; Landau, Amiel-Laviad, Berger, Atzaba-Poria & Auerbach, 2009), as well as parental rejection (e.g., verbal abuse and negative emotion directed towards the child; Kim & Yoo, 2013). However, given investigations often focus on all-male samples (e.g., Keown, 2012; Keown & Woodward, 2002; Landau et al., 2009) or have
disproportionately more boys in their samples (e.g., Chang et al.; Chronis et al., 2007; DuPaul et al., 2001; Gau, 2007; Peters & Jackson, 2008; Rogers et al.; Tandon, Tillman, Spitznagel & Luby, 2014; Tripp et al., 2007), or do not investigate child gender differences in results where possible (e.g., Lifford, Harold & Thapar, 2008), it remains unclear whether these associations also hold for girls. There is evidence that it may not. For example, Tung, Li and Lee (2012) investigated the association between less-than-optimal parenting behaviours (harsh punishment and inconsistent discipline) and conduct problems in a sample of 179 five- to ten-year old children. Results demonstrated that (i) parental harsh punishment was positively related to antisocial behaviour in children, and (ii) inconsistent discipline from parents was positively associated with antisocial behaviour and rule-breaking behaviours in children. These relationships, however, were significant for boys but not girls.

Although the findings of the Tung et al. (2012) investigation suggest that child gender impacts the relationship between parenting practices and externalizing behaviours, the cross-sectional design of the study did not permit an examination of potential prospective or predictive relationships. This highlights a common issue within this area of research. Investigations tend to focus on the impact of child gender on the relationship between parenting and externalizing behaviours cross-sectionally (e.g., Tung et al.) thus precluding the examination of causal relationships; or alternatively, have investigated predictive relationships yet omitted an examination of how these relationships might differ between girls and boys (e.g., Keenan et al., 2010;
Exploring gender differences in ED prevalence

Keown, 2012; Lifford et al., 2008). Further, it is unclear how these relationships might differ for other EDs.

The Pittsburgh Girls Study (PGS), a longitudinal community-based investigation of 2,451 girls recruited between the ages of five- and eight-years (Keenan et al., 2010), has examined prospective relationships, however only in girls. A key aim of the PGS was to examine developmental models of CD as well as other psychiatric disorders. The PGS examined the prospective, bi-directional relationships between girls’ conduct problems and parental displays of harsh punishment and parental warmth. Bi-directional relationships were found whereby harsh punishment and low parental warmth at time one predicted increases in girls conduct problems at time two (12-months later), while girls conduct problems at time one was predictive of parental harsh discipline and lower levels of parental warmth at time two. Despite both directions being significant, a stronger relationship was found for girls’ conduct problems at time one predicting higher harsh and lower warm parenting at time two, but the results cannot be generalised to boys.

There have been similar investigations into the longitudinal association between parenting and child ADHD, however child gender differences have usually not been examined (e.g., Keown, 2012; Lifford et al., 2008). Less-recent studies provide mixed results regarding parenting as a predictor of child ADHD, with some supporting the prospective link (e.g., Carlson, Jacobvitz & Sroufe, 1995) and others not (e.g., Wakschlag & Hans, 1999). More-recent research is beginning to demonstrate relationships in need of further exploration. For example, Lifford et al. (2008) investigated the effects of mother- and father-rejection on child’s ADHD symptomology across a 12-
Exploring gender differences in ED prevalence

month period. Findings demonstrated that only father-rejection at time 1 predicted ADHD symptomology at time 2 (12-months later), while higher levels of ADHD symptoms at time 1 were predictive of higher levels of maternal rejection across these time points. Again, child gender differences were not examined in Lifford et al.’s study, thus it is not known whether these relationships differed between sons and daughters. In a similar investigation, Keown found that higher levels of paternal sensitivity and positive regard, and higher levels of maternal positive regard at baseline were predictive with lower levels of ADHD in sons across a two-and-a-half-year period. Further, lower levels of maternal warmth were predictive of higher levels of sons’ ADHD symptoms across the same time frame. Keown also left unanswered the question of how these relationships may differ based on child gender due to the sample consisting of only boys.

The Lifford et al. (2008) and Keown (2012) studies highlight the importance of examining the prospective associations between parenting and child ADHD, with both papers calling for child gender differences to be examined in future research. Several other important considerations arise from these two studies that should inform future work in this area. First, these studies suggest that the influence of both mothers and fathers should be considered. Much of the previous research into these relationships has examined maternal parenting while seemingly ignoring the relationships between paternal parenting and EDs (e.g., Anderson, Hinshaw & Simmel, 1994; Chi & Hinshaw, 2002; Harold et al., 2013; Healey, Flory, Miller & Halperin, 2011; Peters & Jackson, 2009). This is presumably due to the notion that mothers are often the primary caregivers of children and thus are
more involved in parenting. However, Lifford et al. and Keown both
demonstrate that different prospective relationships may exist for maternal
and paternal parenting practices and EDs. Second, Lifford et al.’s
investigation suggests that bi-directional relationships between parenting and
EDs might exist and thus cross-lagged relationships should be investigated in
prospective studies. This second point suggests a bidirectional-effects model
(Bell, 1964; 1968) likely exists, despite suggestions by some (e.g., Burke,
Pardini & Loeber, 2008; Tarver, Daley & Sayal, 2014; Thapar, Cooper, Eyre
& Langley, 2013) that externalising symptoms are more predictive of
parenting behaviour than the reverse possibility.

A directional relationship between parenting and EDs in children has
received additional support from intervention studies. Psychosocial
interventions aimed at improving parenting have been shown to decrease
disruptive behaviours in children (Bor, Sanders & Markie-Dadds, 2002), and
a recent review of evidence-based psychosocial treatments for children and
adolescents with oppositional- and conduct-related problems found support
for many of the reviewed interventions (Eyberg, Nelson & Boggs, 2008). For
example, Niec, Barnett, Prewett and Shanley Chatham (2016) compared both
individual- and group-based Parent-Child Interaction Therapy (PCIT) in a
sample of 81 families with a child with a diagnosis of either ODD or CD.
Parents in both conditions (i) demonstrated significant improvements in
parenting skills, and (ii) reported significant improvements in their children’s
behaviours and adaptive functioning. Even internet-based parent training,
which involves minimal therapist contact, has been shown to assist with child
conduct-related problems, with these effects still being present at 18-month
follow up (Högström, Enebrink, Melin & Ghaderi, 2015). This collection of research provides evidence that parenting may have an influential impact on the development of externalising behaviours in children, however, for the most part, leaves unanswered the question as to whether the influence of parenting on the development of EDs differs for girls and boys. If different associations do exist based on child gender, this could mean that ED treatments that commonly attempt to modify parenting behaviours might not be as effective for both boys and girls.

6.3 Potential child gender differences in levels of less-than-optimal parenting

While it is possible that the association between parenting and EDs is significant for boys but not for girls, an alternate explanation is that boys and girls might receive different levels of the less-than-optimal parenting practices that are associated with EDs. For example, the male preponderance in ED prevalence rates may be due to the different socialisation of sons and daughters by parents. For example, there is considerable evidence that sons receive higher levels of less-than-optimal parenting than daughters (e.g., more negative parenting styles are directed towards sons than daughters; Lloyd & Devine, 2006; sons receive more corporal punishment and higher levels of physical and verbal aggression than daughters; Mahoney et al., 2000; McKee, et al., 2007) and that less-than-optimal parenting is related to ED development (e.g., Keown, 2012; Lifford et al., 2008). As such, boys may be at an increased risk of ED development compared to girls by being exposed to higher levels of less-than-optimal parenting.
Keenan and Shaw’s (1997) socialisation hypothesis provides a framework from which to conceptualise this potential phenomenon. Socialisation is the process by which individuals learn to behave in a way that is consistent with the standards of the society in which they are raised (Grusec & Hastings, 2014; Grusec & Lytton, 1988; Keenan & Shaw), with parents considered important agents of child socialisation through aspects of their parenting practices and feedback on child behaviour. Keenan and Shaw argued that gender differences in externalising behaviours during middle childhood might occur due to societal factors (such as parenting) encouraging girls to express their difficulties in a less externalizing/more internalising manner and encouraging boys to express their difficulties in a more externalizing/less internalising manner (e.g., girls displaying relational aggression and boys displaying physical aggression; Hay, 2007).

Research that demonstrated the relative absence of gender differences in externalizing behaviours during infancy and early childhood was utilised by Keenan and Shaw (1997) to argue that the early socialisation of gender-specific behaviours may account for gender differences in disordered behaviour arising in middle childhood but not earlier. Keenan and Shaw suggested that boys and girls may experience the same underlying vulnerability to psychopathology, however, as internalising behaviours such as shyness, fearfulness and withdrawal are more socially acceptable in girls than are overactive and aggressive behaviours, girls learn to internalise (rather than externalise) their difficulties more so than boys once they learn these societal norms.
The socialisation hypothesis may extend to boys being ‘socialised’ towards EDs (which are in line with traditional masculine gender roles) and girls being ‘socialised’ away from EDs via parenting practices that are related to ED development. Research into the parenting practices directed towards daughters and sons supports this idea. For example, parents tend to display harsher and more negative parenting styles towards sons than daughters (Lloyd & Devine, 2006), and sons often receive less verbal contact and less supportive speech compared to daughters (Leaper, Anderson & Sanders, 1998). Sons are also more likely to receive corporal punishment, and tend to be exposed to higher levels of physical and verbal aggression than daughters (Mahoney, Donnelly, Lewis & Maynard, 2000; McKee, et al., 2007). Sons and daughters also receive different levels of encouragement from parents for certain behaviours and personality characteristics. For example, parents tend to be more encouraging of autonomy and gender-conformity in boys than girls (Fiese & Skillman, 2000; Kane, 2006), and socialise their sons to prefer physical play and their daughters to prefer social play (Lindsey & Mize, 2001). Given that many of these less-than-optimal parenting behaviours that are directed more towards sons than daughters have been linked to the development of EDs (see section 6.2), this may help to explain the male preponderance in ED prevalence.

6.2 Overview of Study 3

The research aim of Study 3 was to examine the potential bidirectional, prospective relationships between parenting and child ADHD symptoms and diagnosis, and how these relationships may differ based on child gender. Using data obtained from wave 1 (children aged 4- to 5-years) through to
wave 5 (children aged 12- to 13-years) of the Longitudinal Study of Australian Children (LSAC), a large representative sample of Australian children and their parents, the parenting dimensions of Angry, Warm, and Consistent Parenting for both mothers and fathers were examined.

The potential prospective relationships between mothers’ and fathers’ Angry, Warm and Consistent Parenting and child ADHD symptoms were modelled using (i) cross-lagged panel analysis and (ii) latent growth curve model. Further, a logistic regression was used to examine if the abovementioned parenting dimensions at wave 1 were predictive of an ADHD diagnosis at wave 3. Structural invariance testing was conducted on each of these analyses to examine if differences in significant relationships existed based on the gender of the child. Further, child gender differences in the levels of the abovementioned parenting dimensions were tested to examine if boys received higher levels of less-than-optimal parenting than girls. The paper was published in the Journal of Abnormal Child Psychology (see Appendix B for the published version of this study).
Chapter 7: Study 3: The influence of child gender on the prospective relationships between parenting and child ADHD

David H. Demmer¹, Francis Puccio³,⁴, Mark A. Stokes¹, Jane A. McGillivray¹ and Merrilyn Hooley¹

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Reference:


The online supplementary material mentioned in Study 3 been included in Appendices D, E, F and G of this thesis.
# Authorship Statement

## Authorship Statement

1. **Details of publication and executive author**

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<tr>
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<td>School of Psychology</td>
<td>Exploring causes of gender differences in the prevalence of childhood externalising disorders</td>
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If there are multiple authors, give a full description of HDR thesis author’s contribution to the publication (for example, how much did you contribute to the conception of the project, the design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)

- Conceptual development of paper, completion of statistical analyses, drafting of manuscript, revisions, principal author.

I declare that the above is an accurate description of my contribution to this paper, and the contributions of other authors are as described below.

**Signature and date:** 14.03.2017

4. **Description of all author contributions**

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<th>Name and affiliation of author</th>
<th>Contribution(s) (for example, conception of the project, design of methodology or experimental protocol, data collection, analysis, drafting the manuscript, revising it critically for important intellectual content, etc.)</th>
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<tr>
<td>David Hilton Demmer</td>
<td>Conceptual development of paper, completion of statistical analyses, drafting of manuscript, revisions, principal author.</td>
</tr>
<tr>
<td>Dr Francis Puccio</td>
<td>Support with statistical analyses, reviewing and feedback for manuscript.</td>
</tr>
<tr>
<td>Dr Mark Stokes</td>
<td>Support with statistical analyses, reviewing and feedback for manuscript.</td>
</tr>
<tr>
<td>Prof Jane McGillivray</td>
<td>Reviewing and feedback for manuscript.</td>
</tr>
<tr>
<td>Merrilyn Hooley</td>
<td>Thesis supervisor, conceptual development of paper, reviewing and feedback for manuscript.</td>
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* If an author or contributor is unavailable or otherwise unable to sign the statement of authorship, the Head of Academic Unit may sign on their behalf, noting the reason for their unavailability, provided there is no evidence to suggest that the person would object to being named as author.

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Abstract

The aims of the current study were to (i) explore the potential bidirectional, prospective relationships between parenting and child ADHD, and (ii) explore whether these relationships differed on the basis of child gender. Data were obtained from waves 1 (children aged 4- to 5-years) to 5 (children aged 12- to 13-years) of the Longitudinal Study of Australian Child (LSAC) dataset (child cohort). In order to examine dimensions of both mothers' and fathers' parenting, a subsample of nuclear families with mothers, fathers and children present at all waves was extracted (final sample=1,932; sons=981, daughters=951). Child ADHD measures included the hyperactive-impulsive subscale of the strengths and difficulties questionnaire for symptoms, and parent-report question for diagnosis. Mothers and fathers completed scales on dimensions of Angry, Warm and Consistent Parenting. A cross-lagged panel model demonstrated (i) higher child ADHD symptoms at wave 1 led to a global increase in less-than-optimal parenting at wave 2, and (ii) child ADHD symptoms and Angry Parenting shared a prospective, bi-directional relationship (whereby increases in one predicted increases in the other over time) during earlier years of development. Latent growth curve models demonstrated that increases in Angry Parenting across time were significantly predicted by increases in child ADHD symptoms. A logistic regression demonstrated that both mothers’ and fathers’ Angry Parenting at wave 1 significantly predicted an ADHD diagnosis in children at wave 3. No predictive relationships differed between child genders; thus, it appears these prospective pathways are similar for both sons and daughters.

Key words: Childhood psychopathology, ADHD, Externalizing disorder, Parent, Gender
7.1 Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder involving difficulties with attention and/or hyperactive and impulsive behaviours (American Psychiatric Association [APA], 2013). ADHD affects approximately 5% of children worldwide, with prevalence estimates increasing or decreasing based on time period and/or culture considered (Polanczyk, Willcutt, Salum, Kieling & Rohde, 2014). ADHD can have negative impacts on various domains of functioning, including family and peer relationships (Hoza, 2007; Keown & Woodward, 2002) and academic achievement (Barbaresi, Katusic, Colligan, Weaver & Jacobsen, 2007; Reid, Gonzalez, Nordness, Trout & Epstein, 2004), and is also a significant predictor of both concurrent and later psychopathology (Biederman et al., 2006; Burke, Loeber, Lahey & Rathouz, 2005; Meinzer et al., 2016). Therefore, understanding the factors associated with the aetiology and development of ADHD can help inform targets for intervention to assist in lessening these negative consequences.

One factor that has consistently been associated with the development of child ADHD is less-than-optimal parenting. Cross-sectional research has demonstrated that children with a diagnosis of ADHD tend to have parents who are less warm and less involved (Ellis & Nigg, 2009; Tripp, Schaughency, Langlands & Mouat, 2007), less consistent in their punishment (Cussen, Sciberras, Ukoumunne & Efron, 2012), and/or more overprotective (Chang, Chiu, Wu & Gau, 2013) and controlling (Rogers, Wiener, Marton & Tannock, 2009) than the parents of their typically-developing peers. Higher levels of child ADHD symptoms are also related to higher levels of maternal hostility (Harold et al., 2013), parental stress (Graziano, McNamara, Geffken & Reid, 2011), as well as various other aspects of
negative parenting (e.g., poor monitoring/supervision, inconsistent discipline and corporal punishment; Haack, Villodas, McBurnett, Hinshaw & Pfiffner, 2016).

Despite the wealth of investigations into the relationships between various aspects of parenting and child ADHD, several important questions remain unanswered. First, as most previous investigations have been cross-sectional in design, the causal direction of the association between parenting and child ADHD (i.e., whether certain parenting dimensions are predictive of, or an outcome of, child ADHD, or if a bi-directional relationship exists) remains unclear. Second, despite suggestions that child gender may moderate the relationship between parenting and child ADHD (Braza et al., 2015; Johnston & Mash, 2001), this notion is yet to be formally tested (Liffo, Harold & Thapar, 2008).

Two longitudinal studies have explored the prospective relationships between parenting and child ADHD (Keown, 2012; Lifford et al., 2008) in attempts to address the question of causality. The first, Lifford et al., examined the notion of bi-directionality and found different results for mothers and fathers. For mothers, higher levels of child ADHD symptomology predicted higher levels of maternal rejection over a 12-month period, while the reverse relationship was found for fathers, with higher levels of paternal rejection predicting increases child ADHD symptoms in children over the same period. The second, Keown (2012), examined uni-directional relationships only, and again found different relationships for mothers and fathers. Results demonstrated that higher levels of paternal sensitivity and positive regard, and higher levels of maternal positive regard, predicted lower levels of ADHD symptoms in sons across a 2 1/2 year period. Further, lower levels of maternal warmth predicted higher levels of sons’ ADHD symptoms over the same period.
The findings of Lifford et al., (2008) and Keown (2012) contribute to the understanding of the prospective relationship between parenting and child ADHD, however several limitations exist with these investigations that necessitate further work in this area. First, both studies called for investigations utilising larger sample sizes to strengthen the confidence of findings. Second, Keown was limited by investigating only uni-directional relationships (earlier parenting predicting later child ADHD), which precluded the potential identification of alternate, or bi-directional relationships. This is a notable omission given common developmental models of ADHD (e.g., Johnston & Mash, 2001), and the findings of Lifford et al., suggest bi-directional relationships likely exist between parenting and child ADHD. Third, both studies omit child gender as a potential moderator despite the potential importance in examining its effect on the relationship between parenting and child ADHD (Braza et al., 2015; Johnston & Mash).

Gender, specifically male gender, is a known risk factor in the development of ADHD, as demonstrated by the higher prevalence of ADHD in boys when compared to girls (approximately three boys to every one girl in community samples; Erskine et al. 2013; Willcutt, 2012). However, as ADHD research typically involves male-only samples, the way that child gender operates as a risk factor in the development of the disorder remains unclear (Johnston & Mash, 2001). This may have important implications in the treatment of ADHD, particularly if different pathways of disease occur for boys and girls. For example, parent training is an important component of most psychosocial treatments for child ADHD (National Health and Medical Research Council, 2012) based on the notion that parenting impacts child ADHD. Therefore, if parenting is differentially related to ADHD for girls and boys then current interventions may
not be equally effective for both genders if the current ‘one-size-fits-all’ approach to treatment is taken.

Although the notion of whether the prospective relationships between parenting and ADHD differ between daughters and sons is yet to be examined, parallel areas of research suggest a gender difference may exist. For example, it has consistently been demonstrated that parents commonly engage in different parenting behaviours with sons compared to daughters. Sons tend to receive higher levels of authoritarian parenting (e.g., corporal punishment, lack of explanation about punishment, verbal hostility) and less positive parenting (e.g., less displays of warmth, less aware and responsive to cues from the child) than girls (Barnett & Scaramella, 2013; Russell, et al., 1998). Sons also receive fewer displays of emotional understanding (Fivush, Brotman, Buckner & Goodman, 2000), praise, and physical affection than daughters, yet receive higher rates of yelling and smacking (Lloyd & Devine, 2006). Given that (i) many of these less-than-optimal parenting behaviours have been linked to ADHD, and (ii) child gender is related to both parenting and ADHD, it might be that child gender also impacts the relationship between parenting and ADHD. For example, theory (e.g., Keenan & Shaw, 1997) as well as previous research (e.g., Wright et al., 2013) suggests that the socialisation of gender often involves a greater tolerance of internalising behaviours in girls and externalising behaviours in boys by socialising agents (e.g., parents). These socialising influences potentially attenuate the relationship between negative parenting and externalising behaviours in girls, and in boys potentially contribute to the development of a maladaptive cycle of less-than-optimal parenting influencing externalising behaviours, which go on to influence less-than-optimal parenting. It might therefore be expected that less-
than-optimal parenting might have a greater influence on the development of externalising behaviours (such as those demonstrated in ADHD) in boys than in girls.

7.1.2 The current study

The aims of the current study were twofold; first, to explore the potential bidirectional, prospective relationships between parenting dimensions and child ADHD, and second, to explore whether these relationships differed on the basis of child gender, in a large sample of Australian nuclear families. Nuclear families were investigated due to previous research often focusing only on mothers’ parenting. This focus, however, overlooks the important influence of fathers’ parenting on child behaviours. As evidence suggests that child developmental outcomes differ depending on whether maternal or paternal influences are considered (Braza, et al., 2015; Lamb, 2004), and based on previous longitudinal research in this area demonstrating that mothers’ and fathers’ parenting are differentially associated with child ADHD (Keown, 2012; Lifford et al., 2008), it is important to consider both parents in investigations. The current study tested a number of hypotheses: bi-directional relationships were expected based on arguments by common developmental models of ADHD (e.g., Johnston & Mash, 2001). Gender differences in these relationships were also expected based on previous theory (Keenan & Shaw, 1997) that suggests girls might be discouraged from presenting their difficulties in an externalizing manner. It was hypothesized that:

1. Bi-directional, prospective relationships will exist between parenting dimensions and child ADHD symptoms. Specifically, higher levels of less-than-optimal parenting (i.e., higher scores on Angry Parenting, lower
scores on Consistent Parenting, and lower scores on Warm Parenting) will be predictive of, and predicted by, higher levels of ADHD symptoms in boys and girls across time.

2. The predictive relationship between parenting dimensions and child ADHD symptoms will be stronger for boys than girls as measured by chi square change between gender-specific models.

3. Less-than-optimal parenting (i.e., higher scores on Angry Parenting, lower scores on Consistent Parenting, and lower scores on Warm Parenting) at wave 1 will be predictive of an ADHD diagnosis in boys and girls at wave 3.

4. The predictive relationship between parenting dimensions and ADHD diagnosis will be stronger for boys than girls as measured by chi square change between gender-specific models.

7.2 Method

7.2.1 Participants

Data for this study were obtained from waves 1 through 5 of the Longitudinal Study of Australian Children (LSAC). A comprehensive overview of the LSAC sampling design, data collection methods and measures have been described elsewhere (Australian Institute of Family Studies, 2013; Soloff, Lawrence & Johnstone, 2005; Zubrick, Lucas, Westrupp & Nicholson, 2014), thus only a brief outline is provided here. A two-stage cluster sampling design was used to recruit two cohorts, an infant cohort (children 3-19 months of age at wave 1) and a child cohort (children 4-5 years of age at wave 1). First, stratification occurred at the state of residence level, and urban versus rural level. Postcodes (excluding the most remote) were then sampled. Second, all children from sampled postcodes...
who were born between March 2003 and February 2004 (infant cohort), and March 1999 and February 2000 (child cohort), and enrolled in the Australian Medicare Database, which is the most comprehensive database of Australia’s population, were contacted. Final samples were 5,107 for the infant cohort and 4,983 for the child cohort. Only the child cohort was used in the current study due to child ADHD measures being present from wave 1 for this cohort but not for the infant cohort. In order to explore dimensions of both maternal and paternal parenting, a subsample of the original 4,983 child cohort cases was extracted. Cases were included in the current sample if mother, father and child were present, and participated, through all waves, with a final subsample of 1,932; 951 daughters (49.2%) and 981 sons (50.8%).

As causal pathways between non-referred and referred cases are considered fundamentally different, all referred cases (i.e., children with a diagnosis of ADHD) were removed from the cross-lagged path analysis and the latent growth curve models (LGCMs) as per appropriate epidemiological methodology (Rothman, Lash & Greenland, 2008). However, parallel cross-lagged and LGCM analyses were conducted with the full sample (i.e., referred and non-referred cases) with no difference in pathways found between the models. Referred cases were included in the logistic regression as child ADHD diagnosis was the dependent variable in this analysis.

7.2.2 Measures

7.2.2.1 ADHD

Child ADHD symptoms were measured in the LSAC dataset via the strengths and difficulties questionnaire (SDQ; Cronbach’s $\alpha = .88$). Mothers’ SDQ scores were used in the current study as mothers had lower levels of missing data on this
measure compared to fathers. The SDQ is a brief behavioural screening questionnaire for children 4- to 16-years of age, consisting of 25 items covering behavioural and emotional problems, and prosocial behaviours (Goodman, 1997), and contains five subscales. The hyperactivity-inattention subscale is made up of five items (e.g., “Restless, overactive, cannot stay still for long” and “easily distracted, concentration wanders”). Items on the subscale are rated on a three-point index: 0 (not true), 1 (somewhat true), or 2 (certainly true), with a possible range of scores from 0 to 10. Higher scores indicate higher levels of hyperactive-inattentive symptoms. The validity and sensitivity of the SDQ as a screening tool for ADHD symptoms has been well established (Goodman, Ford, Simmons, Gatward & Meltzer, 2000; Stone, Otten, Engels, Vermulst & Janssens, 2010), including in Australian samples (Hawes & Dadds, 2004). For example, Hawes and Dadds used factor analytic techniques to investigate the validity and reliability of the SDQ (parent-report) on an Australian community sample of 1,359 boys and girls 4- to 9-years of age. Moderate to strong internal reliability and stability was found across all subscales, suggesting the SDQ is a valid and reliable measure of both behavioural and emotional symptomology in Australian children.

Child ADHD diagnosis in the LSAC dataset was determined by a single item parent-report question with a yes or no response ("Does child have any of these ongoing problems? ADD or ADHD?"). A second question regarding ADHD medication use ("Has your child ever taken any medication for attention deficit disorder or ADHD?") was used in the current study to validate the response (83% of parents who reported ‘yes’ to ADD or ADHD as an ongoing problem also
reported their children had been prescribed ADD or ADHD medication at wave 5).

7.2.2.2 Parenting dimensions

The parenting dimensions extracted from the LSAC dataset were Angry Parenting, Warm Parenting and Consistent Parenting. These dimensions have previously demonstrated associations with child ADHD (angry: Keown & Woodward, 2002; warm: Chang et al., 2013; Keown, 2012; consistent: Ellis & Nigg, 2009). The construct validity and measurement quality of these scales, as they relate to the LSAC dataset, have been published in a technical paper (Zubrick et al., 2014). For the current study, very good internal consistencies were found for all scales (mothers’ Angry Parenting $\alpha=.86$, fathers’ Angry Parenting $\alpha=.85$; mothers’ Consistent Parenting $\alpha=.88$, fathers’ Consistent Parenting $\alpha=.86$; mothers’ Warm Parenting $\alpha=.88$; fathers’ Warm Parenting $\alpha=.89$). Each scale contained five items with responses provided on a 5-point scale (1 = never/almost never, 2 = rarely, 3 = sometimes, 4 = often, 5 = always/almost always). Both Angry Parenting and Consistent Parenting were measured using items from scales in the National Longitudinal Survey of Children and Youth (NLSYC; Statistics Canada, 2000), while Warm Parenting was measured using a modified scale from the Childrearing Questionnaire (Patterson & Sanson, 1999).

Angry Parenting measured parents’ use of aversive or harsh discipline via items regarding feelings of anger or frustration towards the child, as well as emotional reactivity (e.g., “How often are you angry when you punish this child?”), with higher scores on this measure representing less praise, more disapproval, and more negative emotions directed towards the child. Parents of children with ADHD have been demonstrated to be more angry and mean in their
disciplinary encounters with their children than parents of non-ADHD children (Keown & Woodward, 2002). Further, harsh discipline and/or hostility directed towards children has been shown to influence the development and maintenance of behavioural problems during childhood (Chang, Schwartz, Dodge & McBride-Chang, 2003), while decreases in these parenting behaviours leads to positive change in child behaviour (Sanders, Gooley & Nicholson, 2000).

Warm Parenting measured the amount of warmth and affection displayed towards the child (e.g., “How often do you hug or hold this child for no particular reason?”), with higher scores on this measure representing more displays of warmth and affection. Children with ADHD regularly receive less warmth (Chang, Chiu, Wu & Gau, 2013), with lower levels of parental warmth predicting poorer developmental outcomes for children (Davidov & Grusec, 2006) and substance use disorders in children with ADHD once they reach adolescence (Tandon, Tillman, Spitznagel & Luby, 2014).

Consistent Parenting measured the setting and consistent application of age-appropriate rules and expectations (e.g., “How often does this child get away with things that you feel should have been punished?”). Higher scores on this measure represented the more consistent setting and application of rules and expectations. Inconsistent parenting has been shown to strongly contribute to children’s problem behaviours, and is one of the key areas addressed in behavioural family interventions (Sanders et al., 2000).

7.2.3 Statistical methods

In order to conduct a comprehensive investigation, a series of analyses were selected and conducted to test the hypotheses of the current study and to address different aspects of prospective relationships between parenting and (i) child
ADHD symptoms, and (ii) child ADHD diagnosis. A cross-lagged panel model, two latent growth curve models (LGCMs), as well as a logistic regression, were used to test the hypotheses. All analyses were performed using Mplus version 7.2 and SPSS version 23. Standard cut-offs for fit indices were used: CFI and TLI >.95 for excellent fit and >.90 for acceptable fit and RMSEA <.05 for good fit (Browne & Cudeck, 1993; MacCallum, Browne & Sugawara, 1996; Steiger, 1989). Socio-economic status (SES) was controlled for within all analyses as SES has been shown to be an important associate for both parenting (Friedson, 2016; Pinderhughes, Dodge, Bates, Pettit & Zelli, 2000) and child ADHD (Russell, Ford, Williams & Russell, 2015). The SES variable was a single continuous score calculated based on (i) combined family income, (ii) educational attained of both parents, and (iii) parents’ occupational status. This score therefore represented the social and economic resources available to the family. Rates of missing data for all variables ranged from approximately 0.1% to 8.3% for individual variables across waves. Dimensions of fathers’ parenting generally had the highest rates of missing data across all waves. Missing data were imputed using multiple imputation in MPLUS.

7.2.3.2 Cross-lag panel model

A cross-lagged panel analysis using continuous manifest variables was conducted to test Hypothesis 1. Bi-directional effects examine the reciprocal relationship between two constructs measured across time, and addresses whether a particular construct, measured at a particular time point, is predictive of change in another variable at a later time point. In our model, variance in each of the variables of interest (parenting dimensions and child ADHD symptoms) was predicted from two main sources: autoregressive paths and cross-lagged paths.
The autoregressive paths represent the effect of the construct on itself across time (e.g., child ADHD symptoms at time 1 predicting child ADHD symptoms at time 2; Selig & Little, 2012). The inclusion of autoregressive paths minimizes bias in the estimation of cross-lagged paths (Cole & Maxwell, 2003; Gollob & Reichardt, 1987; Selig & Little). The cross-lagged paths represent the relationships of interest (e.g., the variance in child ADHD at time 2 that is predicted by parenting dimensions at time 2). Given the inclusion of the autoregressive paths, the only variance available for prediction by the cross-lagged paths is the residual variance in the outcome variable, thus providing a more sensitive analysis.

7.2.3.3 Latent growth curve models

Latent growth curve models were also used to test Hypothesis 1. LGCMs assess whether initial levels of one variable (known as the intercept), or the change trajectory (known as the slope) in that variable over time, predicts the change trajectory (slope) of another variable over time. The value of the LGCM is that it establishes how different responses emerge across time, accounting for how earlier decisions influence later outcomes. Further, this enables an understanding of how subtle differences at a given time point may lead to considerable differences in outcome (i.e., sensitive dependence upon initial conditions). Two separate LGCMs were constructed to assess the relationships between the intercept and slope of a predictor variable on the slope of an outcome variable. In the first LGCM, the intercepts and slopes of mothers' and fathers' Consistent, Angry and Warm Parenting were predictors for the slope of child ADHD symptoms. In the second LGCM, the intercept and slope of child ADHD symptoms were the predictor variables, with the slopes of mothers' and fathers' Consistent, Angry and Warm Parenting as outcomes.
7.2.3.4 Logistic regression

A logistic regression analysis was used to test Hypothesis 3, and examined if mothers' and fathers' Consistent, Angry and/or Warm Parenting at Wave 1 predicted a child ADHD diagnosis at wave three. Wave 1 was selected as the prediction wave as the cross-lagged model demonstrated that this was the time point where parenting and child ADHD symptoms were most related. Wave 3 was selected because children at this age (8- and 9-years of age) are at the average age of child ADHD diagnosis (Kessler, Berglund, Demler, Jin, Merikangas & Walters, 2005).

7.2.3.5 Structural invariance testing

Structural invariance testing was conducted for the cross-lag panel model and LGCMs to test Hypothesis 2, and also for the logistic regression to test Hypothesis 4 (except where statistical significance was not found in the relationships of interest), to determine whether the predictive relationships in these models were equivalent across child genders. First, an unconstrained model was tested for boys and girls separately, in which parameters were free to vary. Second, the model was constrained whereby the regression paths for each gender were constrained to be equal. The paths for both models were then compared. Structural invariance (i.e., the models were different based on child gender) was determined if Delta chi-square ($\Delta \chi^2$) significantly differed from zero ($p<.05$).

7.3 Results

7.3.1 Preliminary analyses

Table 1 presents the means and standard deviations (SDs) of parenting dimensions and child ADHD symptoms for non-referred and referred children separately, and summarises the results of t-tests (significant differences and
Cohen’s D effect sizes) comparing non-referred and referred children on parenting dimensions and child ADHD symptoms. Table 2 presents the means and SDs of parenting dimensions and child ADHD symptoms for girls and boys separately, and summarises the results of t-tests (significant differences and Cohen’s D effect sizes) comparing girls and boys on parenting dimensions and child ADHD symptoms. Mothers' and fathers’ Angry Parenting was higher in (i) referred children compared to non-referred children and (ii) non-referred boys compared to non-referred girls across all five waves. From wave 3, referred children received significantly less Warm Parenting from their mothers than non-referred children. This same difference was found for fathers’ Warm Parenting in waves 1 and 3. Mothers' Warm Parenting was higher for daughters than for sons in waves 4 and 5, and fathers' Warm Parenting were higher for daughters than sons from wave 2 onwards. Referred children had significantly higher ADHD symptoms than non-referred children across all waves (see Table 1). Further, non-referred boys had significantly higher child ADHD symptoms than non-referred girls across all waves (see Table 2).
Table 1. Means and standard deviations for, and significant differences and effect sizes (Cohen's D) between, non-referred and referred children across waves

<table>
<thead>
<tr>
<th></th>
<th>Non-referred children Mean (SD)</th>
<th>Referred children Mean (SD) [Cohen's D effect size for non-referred versus referred children t-test]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wave 1</td>
<td>Wave 2</td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angry parenting</td>
<td>2.15</td>
<td>2.13</td>
</tr>
<tr>
<td></td>
<td>(0.57)</td>
<td>(0.55)</td>
</tr>
<tr>
<td>Father</td>
<td>2.26</td>
<td>2.06</td>
</tr>
<tr>
<td>Angry parenting</td>
<td>(0.59)</td>
<td>(0.58)</td>
</tr>
<tr>
<td>Warm parenting</td>
<td>4.42</td>
<td>4.43</td>
</tr>
<tr>
<td></td>
<td>(0.43)</td>
<td>(0.47)</td>
</tr>
<tr>
<td>Man-2</td>
<td>4.07</td>
<td>4.13</td>
</tr>
<tr>
<td>Warm parenting</td>
<td>(0.53)</td>
<td>(0.59)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>4.20</td>
<td>4.27</td>
</tr>
<tr>
<td>consistent parenting</td>
<td>(0.61)</td>
<td>(0.55)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father</td>
<td>4.04</td>
<td>4.15</td>
</tr>
<tr>
<td>consistent parenting</td>
<td>(0.65)</td>
<td>(0.63)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD symptoms</td>
<td>3.14</td>
<td>2.92</td>
</tr>
<tr>
<td></td>
<td>(2.20)</td>
<td>(2.15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: t-tests compared non-referred children to referred children for wave and parenting dimension. E.g., Wave 1 mother angry parenting for non-referred children compared to Wave 1 mother angry parenting for referred children. Significances and effect sizes relate to these comparisons.

*p<.05
**p<.01
***p<.001
ns=t-test non-significant therefore no effect size calcul
Boys  Girls across waves

Rates of ADHD diagnosis, significant differences and effect size (phi), for girls and boys

Table 3

Table 3 presents the rates of ADHD diagnosis across waves for girls and boys, as well as significant differences and effect sizes. Across all waves, boys were significantly more likely than girls to have an ADHD diagnosis.

Table 3

Rates of ADHD diagnosis, significant differences and effect size (phi), for girls and boys across waves

<table>
<thead>
<tr>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
<th>Wave 4</th>
<th>Wave 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Girls</td>
<td>0 (0.00%)</td>
<td>3 (0.31%)</td>
<td>3 (0.31%)</td>
<td>5 (0.52%)</td>
</tr>
<tr>
<td>Boys</td>
<td>10***</td>
<td>17**</td>
<td>34***</td>
<td>35***</td>
</tr>
</tbody>
</table>

Note: t-tests compared girls to boys for Wave and parenting dimension. E.g., Wave 1 mother angry parenting for boys. Significances and effect sizes relate to these comparisons.

*p<.05
**p<.01
***p<.001

ns=t-test non-significant therefore no effect size calculated.
### 7.3.2 Cross-lagged panel model

First, a baseline control model was run containing only the autoregressive pathways: $\chi^2(571, N=1,932) = 2464.38$. The pathways of interest were then added to the model (i.e., the prospective, bidirectional pathways between parenting dimensions and child ADHD symptoms): $\chi^2(523, N=1,932) = 2157.99$. The addition of the pathways of interest significantly improved the model $\chi^2(48, N=1,932) = 306.39$, $p<.001$, demonstrating that the pathways of interest were important additions to the model and predicted significant variance in the model over and above the control/autoregressive model.

Good fit for the cross-lagged model examining the bi-directional effects of parenting dimensions and child ADHD was found (RMSEA=.04, CFI=.96, TFI=.94). For simplicity, Figure 1 presents just the significant findings regarding the relationships of interest (i.e., the bi-directional prospective relationships between parenting dimensions and child ADHD symptoms) as well as the significant autoregressive pathways of the connecting time points. The full table of results is available by request from the corresponding author. Overall, mothers' and fathers' Angry Parenting and child ADHD symptoms demonstrated a prospective, bi-directional relationship; however, this was only significant in the early years (ages 4- to 7-years). Further, this relationship appeared more continuous over time for fathers than mothers. In terms of uni-directional effects, higher levels of child ADHD symptoms predicted higher levels of mothers' and
Exploring gender differences in ED prevalence

fathers' Angry Parenting throughout all waves. In addition, higher levels of child ADHD symptoms at wave 1 predicted decreases in Warm Parenting at wave 2 for fathers and at wave 3 for both mothers and fathers. Higher child ADHD symptoms at preceding waves also predicted less Consistent Parenting at wave four for mothers and wave five for fathers. Increases in mothers' Consistent Parenting at wave three also predicted decreases in child ADHD symptoms at wave four. Structural invariance testing indicated the cross-lag model did not differ based on the gender of the child ($\Delta \chi^2 = p > .05$).
Figure 1. Results of the cross-lagged model. MCon = Mothers consistent parenting, MWar = Mothers warm parenting, MANg = Mothers angry parenting, FCon = Fathers consistent parenting, FWar = Fathers warm parenting, FAn = Fathers angry parenting, ADHD = ADHD scores as measured on SDQ, W = Wave.
7.3.3 Latent growth curve models

Two separate LGCMs were conducted. The paths specified in the LGCMs were identical to the cross-lagged path model however also included the intercept and slopes for ADHD symptoms and each parenting dimension for both mothers and fathers. Model one, where the intercept and slope of mothers' and fathers' Consistent, Angry and Warm Parenting were predictors, demonstrated good fit (RMSEA=.02, CFI=.99, TFI=.99). Model one results demonstrated that no parenting dimension significantly predicted change in child ADHD symptoms over time. Due to this null finding, no invariance testing for child gender was conducted. Model two, where the intercept and slope of child ADHD symptoms were the predictors of the slope of mothers' and fathers' Consistent, Angry and Warm Parenting were outcomes, also demonstrated good fit (RMSEA=.02, CFI=.99, TFI=.99). Model two results indicated that increases in child ADHD symptoms over time significantly predicted increases in both mothers' and fathers' Angry Parenting, as well as decreases in mothers’ Warm Parenting, over time. Child ADHD did not significantly predict any other parenting dimensions. Structural invariance testing demonstrated these relationships were not significantly different based on the gender of the child ($\Delta \chi^2 = p > .05$).

7.3.4 Logistic Regression

The results of the logistic regression demonstrated that mothers' and fathers' Angry Parenting at wave one significantly predicted a child ADHD diagnosis at wave three. No other parenting styles were significant predictors of a child ADHD diagnosis. Structural invariance testing demonstrated these relationships were not significantly different based on the gender of the child ($\Delta \chi^2 = p > .05$). The full table of results can be found in the online supplementary material.
7.4 Discussion

The aim of the current study was to explore the potentially bidirectional prospective relationships between parenting and child ADHD, and to examine if these relationships varied as a function of child gender. Partial support was found for hypothesis one, that predicted bi-directional prospective relationships between less-than-optimal parenting and child ADHD symptoms, with significant prospective, bi-directional relationships found between mothers' and fathers' Angry Parenting and child ADHD symptoms in our cross-lagged panel models, however these bi-directional relationships appeared confined to early years (children aged 4- to 7-years) and were more continuous for fathers than for mothers. Although Angry Parenting no longer predicted child ADHD symptoms from wave three, higher child ADHD symptoms continued to predict higher levels of Angry Parenting throughout all waves in a uni-directional manner. Higher child ADHD symptoms at wave 1 (children age 4- and 5-years) also appeared to lead to an overall increase in less-than-optimal parenting (i.e., higher anger, lower warmth and lower consistency) when children were 6- and 7-years and 10- and 11-years of age, however these patterns were not identical. Child ADHD symptoms also predicted the overall increase in Angry Parenting across all waves, as evidenced in LGCM two. Hypothesis two, that the bi-directional prospective relationships between less-than-optimal parenting and child ADHD symptoms would be stronger for sons than daughters, was not supported as the modelled relationships did not differ as a function of child gender.

Partial support was also found for hypothesis three that predicted less-than-optimal parenting (i.e., higher scores on Angry Parenting, lower scores on Consistent Parenting, and lower scores on Warm Parenting) at wave one would be
predictive of a child ADHD diagnosis at wave three. Only mothers’ Angry Parenting and fathers’ Angry Parenting at wave one were predictive of a child’s ADHD diagnosis at wave three. Hypothesis four was not supported as these predictive relationships between parenting and children’s’ ADHD diagnosis did not differ between sons and daughters.

Mothers and fathers in the current sample appear similar to existing literature in regards to their parenting of sons and daughters. For example, the temporal stability of the autoregressive paths for parenting dimensions in the cross-lagged panel model demonstrate that parenting remains somewhat stable over time (Holden & Miller, 1999; Landry, Smith, Swank, Assel & Vellet, 2001). Further, as is commonly found in parenting literature (e.g., Barnett & Scaramella, 2013; Russell et al., 1998), non-referred sons and daughters in the current sample received different levels of parenting dimensions, with sons receiving higher levels of less-than-optimal parenting compared to girls (e.g., lower levels of warmth and higher levels of anger).

7.4.2 Prospective relationships between parenting and child ADHD

The results of our cross-lagged panel model, LGCM two, and logistic regression add to the existing evidence regarding the prospective relationships between parenting and child ADHD. These results provide some support for claims (e.g., Johnston & Mash, 2001) that a bi-directional relationship exists between child behaviour and parenting. Our results, however, suggest this bi-directional relationship is confined to earlier in childhood and may be more consistent for fathers than mothers, while a uni-directional relationship whereby child ADHD predicts certain parenting dimensions (Angry Parenting in the current sample), continues throughout childhood and into early adolescence.
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Therefore, our results also support arguments that child behaviour may actually be a stronger or more consistent predictor of parenting across time than the reverse relationship (Barkley, 1988; Singh, 2003).

As discussed, the most consistent findings that arose from the current analyses relates to the relationship between child ADHD and mothers' and fathers' Angry Parenting. Our measure of Angry Parenting related to parental feelings of anger and annoyance towards the child, the level of praise/approval directed towards their child, and the difficulty parents experienced in managing their child’s behaviours. Our findings that Angry Parenting shares a bi-directional relationship with child ADHD symptoms in early years before converting to a uni-directional prospective relationship (child ADHD predicting angry parenting) in middle childhood, is in line with previous suggestions that parenting may become less influential on ADHD at later stages of development (Carlson, Jacobvitz & Sroufe, 1995; Johnston & Mash, 2001). The continued influence of child ADHD symptoms on Angry Parenting may have flow-on effects in other domains. For example, parent anger and hostility has been linked to children’s perceptions of parental detachment (Domitrovski & Bierman, 2001), the adoption of a more authoritarian parenting approach (Coplan, Hastings, Lagacé-Séguin & Moulton, 2002), parental conflict (Krishnakumar & Buehler, 2000) and the development of depression in adolescence (Kaitainen, Raeikkoenen, Keskivaara & Keltikangas-Jaervinen, 1999). Further, Angry Parenting behaviours are transmitted generationally, influencing the level of hostility children will later display towards their offspring (Scaramella & Conger, 2003). Thus, ADHD treatments that lower ADHD symptoms may assist in also lowering rates of Angry Parenting and the
negative outcomes associated with angry parenting within, and across, generations.

Our results extend the findings of previous longitudinal studies regarding parenting and ADHD behaviours; for example, the studies by Keown (2012) and Lifford et al., (2008), by considering (i) bi-directional relationships, and (ii) invariance in these relationships across child genders. However, some differences are notable. Keown found less-than-optimal parenting (i.e., lower positive regard, sensitivity and warmth) was predictive of higher child ADHD symptoms across children 4- to 7-years of age. Within the same age range in the current sample, we found a bi-directional relationship for Angry Parenting, and uni-directional influences of child ADHD symptoms predicting less-positive parenting (i.e., lower Warm and Consistent Parenting). Further, across a 12-month period, Lifford et al. investigated the cross-lagged relationships between parental rejection and ADHD symptoms in 11- to-12 year old children. Lifford et al. found higher levels of paternal rejection predicted higher child ADHD symptoms, while higher rates of child ADHD symptoms predicted higher maternal rejection. This suggests different relationships exist between parenting and child ADHD symptoms depending on whether mothers or fathers are considered. Comparison of the findings of waves four and five of the current study (where children were identical in age to the Lifford et al. study) supports their finding that ADHD symptoms predict fathers’ Angry Parenting, however extend this same finding to mothers.

Several factors may account for the differences between the present results and those of Lifford et al., (2008). First, with both mothers and fathers included in a single, bi-directional model in the current study, as opposed to separate models.
for mothers and fathers in Lifford et al., less variance was available in each outcome variable for significant prediction in our model (i.e., significant relationships were harder to find). However, given the models in the current study were stringently controlled with autoregressive paths and for SES, our findings were sensitive to predictive relationships. Second, our models extended over a longer period (children aged 4- to 13-years), compared to Lifford et al. (children aged from 11- to 13-years). As such, we were able to examine predictive relationships across a broader range of developmental periods. Further, by the ages of 11- to 13-years it is possible the behaviours of the children in Lifford et al. were well established, with parenting having only an incidental, rather than predictive, effects.

7.4.3 The influence of child gender on the prospective relationships between parenting dimensions and ADHD

Gender differences were found in several aspects of the current results. First, across all waves, boys had significantly higher ADHD symptoms than girls, and were significantly more likely to have a diagnosis of ADHD, consistent with previous findings (Erskine et al., 2013; Willcutt, 2012). Gender differences were also found in relation to parenting, with sons more likely to receive higher levels of angry parenting than daughters from both mothers and fathers across all waves. However, no gender differences were found in the predictive relationships examined. Taken together with the finding that sons had higher ADHD symptoms than daughters across all waves, this suggests boys and girls are not parented differently as a function of their gender, but rather, differential parenting may occur as a function of child behaviours. This idea requires further investigation.
and suggests that future research into the relationship between parenting and gender should consider child behaviour as an influential associate.

No differences between sons and daughters were found in the prospective relationships between parenting and child ADHD in the current study. Thus, although gender differences existed in the mean levels of ADHD symptoms and parenting dimensions, as well as in the rate of diagnosis, it appears ADHD in both boys and girls share the same relationships with mothers’ and fathers’ Angry, Warm and Consistent Parenting. One possibility that may inform future research is that gender differences may arise when comparing boys and girls who receive similar mean levels of parenting and/or have similar levels of ADHD symptoms.

It may be that child gender more closely impacts the relationship between parenting dimensions, other than those investigated here, and child ADHD symptomology (e.g., autonomy support, coercion, control, responsiveness; Holden & Miller, 1999; Skinner, Johnson & Snyder, 2005). Further research is needed. It may also be that child gender impacts the relationship between parenting styles (i.e., the more stable aspects of parenting and the emotional climate in which parenting occur; Darling & Steinberg, 1993) and child ADHD more so than the relationship between parenting dimensions and child ADHD. As parenting dimensions appear to be more influenced by child behaviours than are parenting styles, it may be that parenting styles are more influenced by child gender. For example, the authoritative and/or authoritarian styles may hold significant differences in their relationships to child ADHD based on child gender than do more transient and reactive parenting dimensions.

It has also been suggested that child gender may be a stronger moderator when considering ADHD subtypes rather than ADHD-combined (Bauermeister et
al., 2007). Thus, the current study may have failed to find significant child gender differences given our measure of ADHD assessed the combined subtype (i.e., included questions regarding both the inattentive and hyperactive/impulsive). It may be fruitful for future research to examine ADHD subtypes when exploring child gender differences in prospective relationships between parenting and child ADHD.

7.4.4 Limitations

The authors acknowledge limitations of the current study. It is possible the mother-report measurement of child ADHD symptomology on the SDQ may have overestimated/underestimated symptoms. Despite the established validity and reliability of the SDQ, previous research has demonstrated some inconsistency between SDQ ratings between informants (e.g., mother, father, teacher, self; Stokes, Mellor, Yeow & Hapidzal, 2014). Thus, different relationships may have been found in our models had different ADHD informants been used. Further, ratings of parenting dimensions and child ADHD symptoms relied on self-report and informant-report, respectively. This may have impacted the validity and reliability of ratings compared to a more objective measure of these variables, such as behavioural observation. Identification of the clinical sample was also based on parent report, and although a further question regarding medication use was used in an attempt to verify clinical status, it is not possible to determine reliability of self-reported diagnosis.

There is also Type 1 error potential given the multiple analyses and pathways in the current study. However, confidence that true relationships were found is increased by (i) the relationships being in the expected directions, and (ii) the fact significant results were found despite the comprehensive autoregressive/control
model. In order to examine both maternal and paternal parenting styles, non-nuclear families (e.g., single-parent families, same-sex parents) were excluded from the current analysis, thus precluding generalisation of our findings to non-nuclear families. Unfortunately, limitations existed in the measurement of culture in the current data, and thus it was unable to be included in the analysis. However, it will be important for future studies to consider the role cultural plays in this area (at the least as a control variable), particularly in countries with high cultural variability.

7.4.5 Conclusion

The results of the current study suggest implications for the psychosocial interventions for child ADHD. In particular, they suggest that treatment should occur within a systemic framework; at a minimum, at the child and parent levels; should involve both parents in dual-family households; and that similar interventions may be effective for both boys and girls. Further, in the early childhood years (between 4- to 7-years of age), addressing levels of Angry Parenting may help to lessen ADHD symptoms in children. Ensuring behaviours associated with angry parenting are addressed in parent training programs for child ADHD, particularly given the possible flow-on effects of angry parenting into other domains of family functioning, may be beneficial. Despite the null findings of the current study in terms of the influence of child gender on the relationship between parenting and child ADHD, the strong association in existing literature between child gender and ADHD, and child gender and parenting, suggests effects are likely occurring between these constructs, though possibly not in the specific parenting dimensions investigated in the current study. Continued
investigations are needed into this notion in order to help understand the aetiology and maintenance factors of child ADHD.

**Ethical approval:** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Informed consent:** Informed consent was obtained from all individual participants included in the study.
References


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Chapter 8: Externalising disorder diagnosis and child gender

The differences between the referred and non-referred male:female prevalence ratios in externalising disorders (EDs) suggest that a substantial number of girls with ED presentations are not being referred for assistance and/or are not being given a ED diagnosis when potentially appropriate, and/or, a substantial number of boys without true ED presentations are being referred and diagnosed when potentially inappropriate. To date, these possibilities have received minimal empirical attention, however some evidence does show that (i) boys with ED presentations may be more likely to be referred to healthcare professionals than girls with identical ED presentations (Sciutto, Nolfi & Bluhm, 2004), and (ii) boys are more likely to receive a diagnosis of ED than girls even when presenting with identical symptoms (Bruchmüller, Margraf and Schneider, 2012). This latter finding suggests that clinicians might contribute to the gender differences in ED prevalence via the possibility of gender-biased misdiagnoses or missed diagnoses.

Gender bias in diagnosis is not a new concept, and has been demonstrated for other disorder groups including personality disorders (Skodol & Bender, 2003). For example, Garb (1997) reviewed literature that investigated gender bias in clinical diagnoses and found when women and men present with identical symptomology relating to personality disorders, a female is more likely to receive a diagnosis for disorders such as borderline personality disorder and depression than is a male. Norman (2004) described this phenomenon by referring to the ‘prototypical representation’ of a disorder. In these cases, the prototype of a disorder includes the gender for which the disorder is most prevalent. As borderline personality disorder and depression demonstrate a higher prevalence in
females, females may fit the prototypical representations for these disorders more so than do males, and this may influence diagnostic decisions. The same phenomenon may be occurring for EDs. It is possible that when a boy is seen clinically he may fit the prototypical representation for a ED better than would a girl with identical symptoms (Bruchmüller et al., 2012), and this may increase the chance of a boy receiving an ED diagnosis, and/or decrease the chance of a girl receiving an ED diagnosis.

Exploring this idea, Bruchmüller et al. (2012) investigated the issue of gender bias in the diagnosis of ADHD in a German sample of mental health professionals. They presented 473 psychologists, psychiatrists and social workers vignettes describing cases with various levels of diagnostic criteria (one that met, and three that did not meet, full diagnostic criteria) introduced as either a boy or a girl. Bruchmüller et al. predicted clinicians would be more likely to base their ADHD diagnoses on diagnostic heuristics, which included the gender of the child as well as the most prominent or usual symptoms, rather than on direct reference to diagnostic manuals (e.g., DSM-IV-TR and ICD-10). Two main findings arose from this study: First, in an analysis of clinicians who had made a diagnosis, 20% provided a diagnosis of ADHD when full diagnostic criteria were not met and therefore it was inappropriate to do so (i.e., false positive diagnosis). Interestingly, only 8.2% of clinicians did not provide a diagnosis in cases where a diagnosis was applicable (i.e., false negative diagnosis). This suggests over-diagnosis may be a more predominant issue than under-diagnosis.

Second, Bruchmüller et al. (2012) found boys were twice as likely as girls to receive a false positive diagnosis of ADHD, yet there appeared little difference between boys and girls in false negatives. These findings provide clues as to how
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Clinicians may be influencing the male:female prevalence ratios of EDs, and support the idea that males may be seen as ‘prototypical representations’ of individuals with ADHD, and that this gender bias may permeate the diagnostic considerations of clinicians.

Despite the interesting results demonstrated by the Bruchmüller et al. (2012) study, it remains unclear whether clinicians from countries other than Germany demonstrate similar biases in ADHD diagnosis. There were also several limitations to the Bruchmüller et al. study that invite replication. First, Bruchmüller et al. required participants to provide a diagnosis despite a diagnosis being inappropriate in two of the four vignettes. This likely increased the rates of false positive diagnoses. A more appropriate approach may have been to offer clinicians the option of proving either a (i) diagnosis, or (ii) working hypothesis for the disorder they believed best accounted for the child’s symptoms, along with a confidence rating of their decision. Second, the use of a between-subjects design precluded the opportunity of Bruchmüller et al. to examine individual-level propensities to misdiagnose ADHD.

8.2 Rationale for Study 4

The aim of Study 4 was to examine the influence of child gender on the diagnostic decision-making processes of clinicians when assessing children for psychological disorders by replicating and extending the methodology of Bruchmüller et al. (2012). The study was prepared for publication and is currently under review in the Journal of Clinical Child and Adolescent Psychology.
Chapter 9: Study 4: The influence of child gender on ADHD diagnosis

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Reference:


Additional material relating to this study are included as the following appendices: Letter to organisations (Appendix H), plain language statement (PLS) and consent form for organisations (Appendix I), PLS for participants (Appendix J), online survey time 1 (Appendix K), online survey time 2 (Appendix L), full vignettes 1 – 3, male and female versions (Appendix M), proof of journal submission (Appendix N)
ABSTRACT

Objective: To investigate if child gender influences clinicians’ diagnostic decisions. Method: At time one, fifty-three clinicians of various backgrounds received a case vignette for a boy or girl, describing behaviours that either (i) met ADHD diagnostic criteria with subclinical major depressive disorder (MDD) symptoms, (ii) contained subclinical ADHD and MDD symptoms, or (iii) met MDD diagnostic criteria with subclinical ADHD symptoms. At time two, clinicians received the same vignette for the opposite-sex child. Results: Cross-sectionally, ADHD was appropriately considered in 19/20 (95%) cases where full ADHD criteria were met, and misdiagnosed in 22/33 cases (66.66%) where criteria were not met. MDD was misdiagnosed with 15/16 clinicians failing to diagnose MDD where criteria were met. Similar patterns were found longitudinally. Child gender did not appear to influence clinicians’ decisions. Conclusion: Clinicians do not appear to consider child gender when diagnosing ADHD, however demonstrate a propensity to misdiagnose ADHD and MDD.
9.1 Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a common childhood disorder encompassing symptoms of inattention and/or impulsivity and hyperactivity (American Psychiatric Association [APA], 2013) that affects approximately five percent of the child population (Wittchen et al., 2011). A diagnosis of ADHD is associated with numerous negative consequences for the individual, such as poorer educational (Biederman et al., 2004; Daley & Birchwood, 2010; Loe & Feldman, 2007), social (Bagwell, Molina, Pelham & Hoza, 2001; Hodgens, Cole, & Boldizar, 2000; McQuade & Hoza, 2008), and psychological (Biederman et al., 2006; Ingram, Hechtman, & Morgenstem, 1999) outcomes. An accurate diagnosis of ADHD, where applicable, is therefore crucial to assist those with the disorder (Cormier, 2008). Further, given that ADHD treatment often involves psychopharmacological intervention, an accurate ADHD diagnosis is essential in managing the possible over prescription of potentially harmful medication to children (Shaw, Wagner, Eastwood, & Mitchell, 2003).

Despite the importance of an accurate ADHD diagnosis, discussion continues regarding possible diagnostic inaccuracies and misdiagnosis (Desgranges, Desgranges & Karsky, 1995; Goldman, Genel, Bezman, & Slanetz, 1998; Lilienfeld & Arkowitz, 2013; Partridge, Lucke, & Hall, 2014; Sciutto & Eisenberg, 2007; Shaw et al. 2003). Misdiagnosis includes false positives (i.e., providing an ADHD diagnosis when it is inappropriate to do so) or false negatives (i.e., failing to provide an ADHD diagnosis when the disorder is actually present). Although older research pointed to a misdiagnosis of ADHD, there has been little recent research exploring this issue. For example, Cotuono (1993) examined clients at a community health center who had been referred with an existing
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Diagnosis of ADHD, and found that only 20 of those 92 clients were assigned a primary diagnosis of ADHD following a comprehensive reassessment. While this issue may be influenced by biased referrals (e.g., a tendency for boys to be referred more so than girls; Sciutto, Nolfi, Bluhm, 2004; referrals that suggest the preconceived presence of ADHD; Desgranges, Desgranges & Karsky, 1995), it is important to understand the assessment and diagnostic processes of clinicians and evaluate these for potential inaccuracies.

Several factors have been suggested to influence ADHD misdiagnosis in the clinical setting. Sciutto and Eisenberg (2007) suggest false positives may arise from the misattribution of symptoms to ADHD when they are indicative of an alternative disorder. For example, behaviors consistent with ADHD, such as noncompliance or inattention, may occur due to a different externalizing disorder (e.g., noncompliance symptomatic of oppositional defiant disorder), or internalizing disorder (e.g., inattention symptomatic of major depression), but be attributed to ADHD. This misattribution possibly occurs due to poor adherence to best-practice assessment guidelines (Handler & DuPaul, 2005; Sciutto & Eisenberg), and would lead to misdiagnosis across numerous disorders, resulting in a false positive for ADHD and a false negative for the disorder/s actually present. This could then flow on to the possible misapplication of both psychosocial and pharmacological interventions.

Another factor suggested to influence ADHD diagnoses is child gender, in that boys may be more likely to receive an ADHD diagnosis than girls (Bruchmüller, Margraf & Schneider, 2012; Sciutto & Eisenberg, 2007). Gender is hypothesized to influence diagnosis in two ways: First, as ADHD prevalence is higher in boys than girls, child gender may have become part of an assessment or
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diagnosis heuristic, with the *prototypical representation* of a child with ADHD including ‘being male’ (Bruchmüller et al.). In this way, assessment of diagnostic heuristics that include gender considerations may be associated with false positive diagnoses in boys and false negative diagnoses in girls regardless of symptoms and/or presentation (Bruchmüller et al.). Second, as girls with ADHD tend to present with fewer hyperactive/impulsive symptoms than boys with ADHD, girls so may be less disruptive and therefore overlooked for referral and assessment (Sciutto & Eisenberg).

To explore this possibility, Bruchmüller et al. (2012) recruited a sample of 473 psychologists, psychiatrists and social workers from various German states and presented the clinicians with one of four written case vignettes based on DMS-IV and ICD-10 diagnostic criteria for ADHD. The cases outlined various child presentations, some of which fulfilled ADHD diagnostic criteria and some that did not. Vignettes were presented as either a boy or girl, and a diagnosis was requested. The authors predicted clinicians would be more likely to base their diagnoses on aspects of a diagnostic heuristic (e.g., child gender) rather than direct reference to diagnostic manuals (e.g., DSM-IV-TR and ICD-10), and thus would diagnose ADHD even when it would be inappropriate to do so. These predictions were supported. In their analysis of clinicians who made a diagnosis, 20% made a false positive diagnosis and 8.2% provided false negative diagnoses, representing an over diagnosis of ADHD. Importantly, Bruchmüller et al. found that vignettes involving boys were twice as likely as vignettes involving girls to receive a false positive diagnosis of ADHD, yet there appeared little difference between boys and girls in false negatives. These findings support the idea that child gender is part of the prototypical representation (i.e., heuristic) of an
individual with ADHD, and that this gender bias may permeate the diagnostic decisions of clinicians.

Despite the informative results of Bruchmüller et al. (2012), several limitations within the study invite its replication and expansion. First, Bruchmüller et al. required a diagnosis from participants despite the fact that in two of the four vignettes no diagnosis was applicable; thus forcing a false positive diagnosis in two of the four cases. An alternative approach may have been to offer clinicians the option of proving a working hypothesis (WH) for the disorder they believed best accounted for the child’s symptoms. In addition, further valuable information may have been provided by (i) an indication of the clinician’s level of confidence in their decision, and (ii) identification of salient symptoms on which the diagnostic decision was based. Second, a within-subjects design not only would have improved sensitivity to explore gender differences in diagnosis, it would have provided the opportunity to explore individual-level propensities to misdiagnose ADHD.

In line with the potential of the prototypical representation of a child with ADHD (an externalising disorder) including ‘being male’ (Bruchmüller et al. 2012), a similar process may occur with girls whereby the prototypical representation of a child with an internalising disorder may include ‘being female’. For example, many internalizing disorders (e.g., Major Depressive Disorders; MDD; minor depression; Ezpeleta, de la Osa & Domenech, 2014) have a higher prevalence in girls than boys, and in females across most age groups (Bebbington, et al. 2003). It is possible that the diagnosis of MDD is influenced by child gender. Although some older research exists that demonstrates women are more likely to be diagnosed with MDD than men (regardless of whether a
diagnosis is applicable or not; Potts, Burnam & Wells, 1991), no such investigations have been conducted exploring this possibility in children.

The aim of the current study was to replicate the findings of Bruchmüller et al., (2012) while implementing modifications to address the limitations outlined above. Symptoms for a second disorder (Major Depressive Disorder; MDD) were also included in the vignettes for two reasons. First, it might be that the prototypical representation of a child with MDD includes ‘being female’, and second, MDD can share a similar presentation to ADHD depending on the constellation of symptoms despite MDD being classified as an internalising disorder. Therefore, the inclusion of MDD sought to examine (i) whether any patterns of misdiagnosis occur for MDD whereby this disorder is diagnosed or considered more commonly for girls than boys, and (ii) whether ADHD is diagnosed or suggested even when symptoms might actually be attributable to MDD. It was hypothesized that:

1. There would be a higher proportion of ADHD diagnoses in vignettes involving boys than girls.
2. There will be a higher proportion of MDD diagnoses in vignettes involving girls than boys.

9.2 Method

9.2.1 Participants

Participants were health care clinicians who are potentially involved in the assessment, diagnosis and/or treatment of ADHD. These included psychologists, psychiatrists, paediatricians and general practitioners (GPs), recruited via their professional associations in Australia: the Australian Psychological Society (APS), the Royal Australasian College of Physicians (RACP), the Royal
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Australian and New Zealand College of Psychiatrists (RANZCP) and the Royal Australian College of General Practitioners (RACGP) for psychologists, paediatricians, psychiatrists and GPs respectively.

The sample consisted of 35 psychologists, 14 paediatricians and four psychiatrists (N=53; 13 males and 40 females); no GPs participated. Ages ranged between 23 to 79 years (M=42; SD=14.2). The years practicing in their respective professions ranged from less than one to 42 (M=12.2; SD=2.4). The majority of clinicians either had a low (0-25% of current case load; 40%) or high (76-100% of current case load; 41%) current caseload of child and adolescent clients.

Clinicians expressed a moderate degree of confidence working with child and adolescent clients (M=7.0; SD=2.4) using a 10-point likert scale (higher scores representing higher confidence).

9.2.2 Materials

9.2.2.1 Vignettes

Three written case vignettes were designed based on DSM 5 diagnostic criteria for ADHD combined-type (ADHD-C) and major depressive disorder (MDD), with the number of symptoms (and thus possibility of a diagnosis) differing between vignettes. Two versions of each vignette were created; one presented a 10-year-old girl (Sarah), the other a 10-year-old boy (Simon). A team of clinical psychologists with a specialisation in the area of child and adolescent psychology reviewed the vignettes, with alterations made based on their expert feedback.

Vignette A – Diagnosable ADHD-C, subclinical MDD

Vignette A met full DSM 5 criteria for ADHD-C as follows: Criteria A) six or more inattentive symptoms plus six or more hyperactive and impulsive symptoms.
Criteria B) Symptoms were present prior 12-years-of-age. Criteria C) several symptoms were present across two or more settings. Criteria D) Symptoms interfered with functioning. Criteria E) Symptoms were not due to another mental disorder. Vignette A also contained MDD symptoms, however these did not meet full diagnostic criteria (two symptoms missing).

Vignette B – Subclinical ADHD and subclinical MDD

Vignette B contained only five inattentive symptoms and five hyperactive/impulsive symptoms. Vignette B also contained MDD symptoms, however these did not meet full diagnostic criteria (one symptom missing). Therefore, no diagnosis was applicable in Vignette B.

Vignette C – Subclinical ADHD, diagnosable MDD

Vignette C contained four inattentive symptoms and four hyperactive/impulsive symptoms. Therefore, an ADHD diagnosis was not applicable in vignette C. Full MDD diagnostic criteria were met, therefore it was appropriate to provide a diagnosis of MDD in vignette C.

9.2.3 Procedure

Clinicians complete two online surveys approximately two weeks apart. The time one survey collected clinicians’ demographic information and randomly presented one of the six vignettes (i.e., vignette A, B, or C for ‘Sarah’ or ‘Simon’). Clinicians were requested to read the vignette before responding to the following questions: (i) Did they believe the child met a diagnosis for a DSM 5 psychological disorder? If ‘Yes’ clinicians were asked to provide the diagnosis. If ‘No’ clinicians were asked to indicate their working hypothesis (WH; i.e., what disorder did they think the child was most likely to have based on the vignette). (ii) List the most salient/important features of the child’s presentation on which
they based their diagnosis or WH, (iii) Indicate any comorbid DSM 5 disorders, or features of other DSM 5 disorders, they believed were present, and (iv) Rate their confidence in their diagnosis or WH on a 10 point likert scale where higher scores represented greater confidence. Two weeks later, clinicians received the time two survey. The vignette provided was the same as at time one but with the opposite sex child. For example, if clinicians received vignette A (boy) at time one, that clinicians received vignette A (girl) at time two.

9.3 Results

A power analysis was conducted using g*power using an alpha level of 0.05 which estimated that a sample size of 66 participants would be able to detect a moderate effect (Cohen, 1992). The moderate effect was estimated based on the findings of Bruchmüller et al. (2012). Due to the small sample size and low power, planned analyses designed to explore data were limited. Thus, the data are presented and described with statistical analyses undertaken where possible. Table 1 outlines the diagnostic decisions made by clinicians at time one. In vignettes where full diagnostic criteria for a diagnosis were evident, ADHD was appropriately considered in 19/20 (95%) cases in vignette A, and MDD was appropriately considered in 1/16 (6%) cases in vignette C. A chi-square comparison of proportions showed that this difference was significant ($\chi^2(1)=27.710$, $p<.001$). In vignettes where diagnostic criteria were not met, ADHD was misdiagnosed in 22/33 cases (66.66%) in vignettes B and C, and MDD in 1/37 (2.77%) in vignettes A and B; this difference was significant ($\chi^2(1)=34.091$, $p<.001$). Clinicians reported a moderate level of confidence in their diagnostic decisions at time one ($M=7.0$; $SD=2.01$). Confidence remained stable across vignettes; vignette A ($M=6.9$; $SD=2.2$), vignette B ($M=6.9$;
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An independent samples t-test was conducted to examine differences in confidence when making a diagnostic decision for boys ($M=7.0$, $SD=2.08$) and girls ($M=6.97$, $SD=2.13$) at time one. No significant difference was found ($t(51)=-0.06$, $p>.05$). In regards to the symptoms that clinicians based their diagnostic decisions on, 79.2% considered hyperactive/impulsive symptoms when making their decision and 84.9% considered inattentive symptoms.
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Table 1  
*Diagnostic responses to vignettes at time one*

<table>
<thead>
<tr>
<th>Vignette (gender)</th>
<th>n</th>
<th>ADHD diagnosis</th>
<th>ADHD WH</th>
<th>MDD diagnosis</th>
<th>MDD WH</th>
<th>Other response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
</tr>
<tr>
<td>A (Girl)</td>
<td>12</td>
<td>10 (83.5%)</td>
<td>2 (16.5%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>A (Boy)</td>
<td>8</td>
<td>5 (62.5%)</td>
<td>2 (25%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (12.5%)</td>
</tr>
<tr>
<td>B (Girl)</td>
<td>8</td>
<td>3 (37.5%)</td>
<td>2 (25%)</td>
<td>1 (12.5%)</td>
<td>0 (0%)</td>
<td>2 (25%)</td>
</tr>
<tr>
<td>B (Boy)</td>
<td>9</td>
<td>7 (77.78%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (11.11%)</td>
<td>1 (11.11%)</td>
</tr>
<tr>
<td>C (Girl)</td>
<td>9</td>
<td>7 (77.78%)</td>
<td>2 (22.2%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>C (Boy)</td>
<td>7</td>
<td>5 (71.42%)</td>
<td>0 (0%)</td>
<td>1 (14.29%)</td>
<td>0 (0%)</td>
<td>1 (14.29%)</td>
</tr>
</tbody>
</table>

WH=Working Hypothesis
Other diagnoses/WHs included: Diagnosis of Autism, developmental delay; WH of intellectual disability, bipolar, potential traumatic event
9.3.2 ADHD

In order to test hypothesis one, that there would be a higher proportion of ADHD diagnoses in vignettes involving boys than girls, a chi-square comparisons of proportions test was used. Clinicians were no more likely to provide an ADHD diagnosis for boys than girls in vignette A ($X^2(1)=1.07, p>.05$), vignette B ($X^2(1)=2.67, p>.05$), or vignette C ($X^2(1)=0.08, p>.05$). Overall, clinicians were just as likely to provide an ADHD diagnosis in vignette A (the diagnosable case of ADHD) compared to vignettes B and C (the non-diagnosable cases of ADHD; $X^2(1)=0.403, p>.05$). A one-way ANOVA revealed no difference in confidence in diagnostic decisions between clinicians who provided an ADHD diagnosis for the three vignettes (i.e., clinicians who appropriately considered ADHD as either a diagnosis or WH in vignette A [$M=6.95; SD=2.24$] and clinicians who inappropriately provided a diagnosis in vignettes B [$M=7.8; SD=1.75$] and C [$M=7.21; SD=2.08$]) [$F(2,38) = 0.76, p>=.05$].

9.3.3 MDD

MDD was rarely considered in cases where full diagnostic criteria were met (i.e., false negatives), with only 1/16 (6%) clinicians considering a diagnosis for MDD where it was appropriate. MDD was misdiagnosed by 1/37 (2%) clinicians where full criteria were not met. Due to small cell sizes, no analyses were possible; therefore hypothesis two could not be tested.

9.3.4 Time Two

Of the 53 clinicians who participated at time one, 31 completed the time two survey. Ten clinicians changed their diagnostic decisions across vignettes. Of those who provided correct responses at time one 30% changed to incorrect responses at time two, and of those who provided incorrect responses at time one
14% changed to correct responses at time two. Table two outlines the changes in responses from time one to time two, and whether decisions became more or less correct over time. No trends were observable.

Of the 13/31 clinicians who received an ADHD diagnosable vignette at times one and two, four changed their decision at time two. Two provided a diagnosis of ADHD for the female version but an ADHD WH for the male version, and two provided a diagnosis of ADHD for the male version but an ADHD WH for the female version. Of the 11/31 clinicians who received a MDD diagnosable vignette at times one and two, two changed their decisions. One provided a diagnosis of ADHD for the male version but an ADHD WH for the female version, and one provided a diagnosis of ADHD in the female version but a WH of bipolar disorder for the male version.

An independent samples t-test was conducted to examine differences in confidence when making a diagnostic decision for boys (\(M=7.19, SD=1.60\)) and girls (\(M=6.80, SD=1.87\)) at time two. No significant difference was found (\(t(29)=-0.60, p=>.05\)). A paired samples t-test was also conducted to examine differences in confidence between decisions at time one (\(M=70.3, SD=1.92\)) and time two (\(M=7.06, SD=1.67\)). No significant difference was found (\(t(30)=-0.162, p=>.05\)).
Table 2. 
*Changes in diagnostic decisions from time one to time two*

<table>
<thead>
<tr>
<th>Vignette A</th>
<th>Vignette B</th>
<th>Vignette C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time 1</td>
<td>Time 2</td>
<td>Time 1</td>
</tr>
<tr>
<td>ADHD diagnosis</td>
<td>ADHD WH(^a)</td>
<td>ADHD WH</td>
</tr>
<tr>
<td>ADHD diagnosis</td>
<td>ADHD WH(^a)</td>
<td>ADHD diagnosis</td>
</tr>
<tr>
<td>ADHD WH</td>
<td>ADHD diagnosis(^b)</td>
<td>ADHD diagnosis</td>
</tr>
<tr>
<td>ADHD WH</td>
<td>ADHD diagnosis(^b)</td>
<td>Other WH</td>
</tr>
</tbody>
</table>

\(^a\)Became less correct over time  
\(^b\)Became more correct over time  
\(^c\)No change in correctness over time
9.4 Discussion

The aim of the current study was to examine whether clinicians were influenced by child gender when making diagnostic decisions regarding ADHD. It was hypothesized that there would be a higher proportion of ADHD diagnoses or WHs in vignettes involving a boy than involving a girl. Our results did not support this hypothesis. Regardless of child gender, clinicians demonstrated a propensity to diagnose, or at least provide a WH of, ADHD. It was also hypothesized that there would be a higher proportion of MDD diagnoses or WHs in vignettes involving a girl than involving a boy. This hypothesis too was not supported.

The failure to find that child gender influences ADHD diagnostic decisions is inconsistent with the results of Bruchmüller et al. (2012). We offer several possible reasons for the inconsistency. First, it may be that child gender does play a role in the diagnostic decisions made by the clinicians in our study, however our small sample size restricted our ability to explore this thoroughly. Second, our sample involved Australian clinicians compared to German clinicians in the Bruchmüller et al. study. There may be societal or cultural differences that exist between Australia and Germany that affect clinicians’ reliance on child gender when considering an ADHD diagnosis. The between-subjects methodology of Bruchmüller et al. also potentially confounds the influence of sex-of-target effect with the individual differences between participants in their diagnostic decisions. The methods of the current study, which engaged both a between- and within-subjects design, attempted to overcome this by permitting both (i) a sex-of-target effect while also (ii) exploring potential patterns in diagnostic decisions made by participants. Examination of changes in decisions at the individual-participant
level suggests clinicians do not change their diagnostic decisions based on child gender, however should be interpreted within the context of our limited sample size.

The clinicians in the current study demonstrated a clear propensity to diagnose ADHD regardless of child gender and/or the quantity of ADHD symptoms, suggesting false positive diagnoses of ADHD are occurring. While Bruchmüller et al. (2012) suggests this phenomenon may be influenced by the gender of the child, our findings, which show false positives occur equally in boys and girls, are not consistent with this idea. Rather, we suggest that ADHD is possibly primed in the minds of clinicians as they engage in the assessment of child psychological disorders. Clinicians may expect the presence of ADHD given its high prevalence during childhood, or seek to confirm a working hypothesis of ADHD when one or two ADHD-consistent behaviours are present rather than to disprove it. The nature of referrals may also influence this potential issue. It has been suggested that increased public awareness of ADHD has led to families referring children for problem behaviours with the expectation that their child has ADHD (Desgranges, Desgranges & Karsky 1995). If referral information from parents, teachers, or other professionals over-emphasises well-known symptoms consistent with ADHD, and under-emphasises less-well-known symptoms associated with less observable disorders clinicians may unknowingly seek to confirm an ADHD diagnosis rather than disconfirm it, or alternatively may fail to explore alternate diagnostic possibilities.

There is also the suggestion that the externalising nature of ADHD symptoms may play a role in misdiagnosis (Sciutto & Eisenberg, 2007). For example, ADHD may be primed more when a child presents with the hyperactive and/or
impulsive symptoms than when they present with the inattentive symptoms. If this were the case, then it would be expected that clinicians in the current study would nominate *externalising* symptoms more often than *internalising* symptoms when outlining which symptoms they based their diagnostic decisions on. This, however, was not the case. Clinicians in the current sample nominated hyperactive, impulsive and inattentive symptoms at a similar rate.

A strength of our design was the inclusion of a second disorder which permitted us to explore whether potential misdiagnosis of ADHD reflected clinicians’ efforts to protect against potential false negative diagnoses, which would lead vulnerable children to miss out on treatment. It is possible that clinicians err on the side of caution to be overinclusive in their diagnoses rather than risk excluding these vulnerable children from treatment and support protocols. If this were the case, we might expect clinicians would provide false positive diagnoses for children described with subclinical ADHD and MDD equally. This was not the case. While approximately 25% of cases meeting ADHD criteria did not receive a diagnosis from our clinicians, 94% of cases of MDD that met criteria, did not receive an MDD diagnosis. The false positives for ADHD also well outnumbered those for MDD in sub-clinical cases. Whether the misdiagnosis of MDD occurs just in the presence of ADHD symptoms, or as a routine occurrence, requires further investigation.

Taken together, the false positives of ADHD and false negatives of MDD support two notions. First, the notion offered by Sciutto and Eisenberg (2007) that symptoms representative of another disorder may be misattributed to ADHD in cases where one or more classic symptoms of ADHD are present. Where there is overlap between disorders in their diagnostic criteria (e.g., issues with attention...
that can be present for both ADHD and MDD), careful assessment and consideration should be paid to all diagnostic possibilities to avoid inaccurate diagnoses being applied. Second, they support the notion that symptoms of alternate disorders may be minimized or overlooked in the presence of ADHD. Any tendency toward false positive diagnoses would also falsely increase the prevalence of ADHD.

This also highlights important issues for the treatment of childhood disorders. Psychostimulant medication is often a key aspect of ADHD treatment (National Health and Medical Research Council, NHMRC, 2012), and false positive ADHD diagnoses may lead to children being prescribed psychostimulant medication that involve negative side effects such as insomnia, appetite disturbance, stomachache, headaches and/or dizziness (Ahmann et al. 1993). Medication to manage these side effects may then be prescribed while the original difficulties remain potentially untreated.

### 9.4.2 Limitations

The authors acknowledge several limitations to the current study. First, our limited sample size impacted our ability to perform sophisticated statistical analyses restricting the interpretation of the results. Second, carryover effects between time one and time two measures are possible. On average, two weeks separated time one and time two surveys, and it is likely clinicians had some recall of their decisions at time one when completing time two. However, although some clinicians may have remembered their diagnosis or working hypothesis, there was no discernable trend for diagnoses to become more or less accurate from time one to time two.
There are also limitations associated with the use of written case vignettes. Guidelines in the assessment and diagnosis of ADHD suggest a comprehensive assessment including, but not limited to, physical and mental health, cognitive, behavioural, education and psychosocial functioning, as well as the consideration of cultural factors (NHMRC, 2012). Clinicians generally have more information available to them to make diagnostic decisions than was provided by our case vignettes. However, the provision of a working hypothesis sought to overcome this issue. Despite having this option, many clinicians provided a diagnosis and reported a high level of confidence in their diagnostic decisions.

9.4.3 Conclusion

The current study suggests child gender is not taken into account by clinicians in their diagnostic decision making processes regarding ADHD, and that there is a clear propensity for clinicians to diagnose ADHD even when subclinical, and in the presence of diagnosable MDD. Of concern is the relatively high confidence clinicians reported in their diagnostic decisions in the presence of an incorrect diagnosis. Further research is needed in this area to develop a sound understanding of the diagnostic processes of ADHD assessment and the factors leading to false positive diagnoses. Further research is also needed into the possible false negative diagnoses occurring for MDD. These misdiagnoses could have significant impacts on the treatment and recovery of children experiencing a variety of difficulties during childhood if disorders are inappropriately identified or missed.
References


Exploring gender differences in ED prevalence


Chapter 10: General discussion

The gender imbalance in the reported prevalence rates of externalising disorders (EDs) suggest that factors associated with the (i) development of EDs and/or (ii) diagnosis of EDs, might differ between girls and boys. To date, how these factors might differ based on child sex and/or gender has remained underexplored and not well understood (Bruchmüller, Margraf & Schneider, 2012; Gaub & Carlson, 1997; Kann & Hanna, 2000; Reid, et al., 2000). The current thesis has begun to explore this gap in knowledge via two aims: First, it sought to summarise the current state of knowledge regarding the male:female prevalence ratios of attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiant disorder (ODD) during middle childhood in non-referred children. Second, it investigated how (i) parenting, and (ii) diagnosis may differ between boys and girls in the most common ED, ADHD, as possible explanations for the gender imbalance in ADHD prevalence.

These two broad aims were examined via four individual studies. Study 1 was an overview of systematic reviews that summarised the current state of knowledge regarding the male:female prevalence ratios of ADHD, CD and ODD during middle childhood in non-referred children. Given that the results of Study 1 demonstrated that no meta-analysed male:female prevalence ratio of ODD existed at the time of the systematic search, Study 2 provided a meta-analysed male:female prevalence ratio of ODD during middle childhood in non-referred children. Studies 3 and 4, utilizing ADHD as their ED of interest, examined how the social-level factors of (i) parenting, and (ii) diagnosis, might differ between boys and girls as explanations for the gender imbalance in ADHD prevalence. The overarching hypothesis for the investigations undertaken in Studies 3 and 4 was
that the development and diagnosis of ADHD would differ between boys and girls. Only partial support was found for this overarching hypothesis, with the results of Study 3 demonstrating that boys receive higher levels of Angry Parenting than girls, and that this may have contributed to the higher ADHD symptoms in the boys in the study, particularly given the overall association between parenting and child ADHD did not differ based on child gender. The other results of Study 3 that demonstrated the prospective, bi-directional relationships between parenting and child ADHD did not differ based on child gender, and the results of Study 4, did not support this overarching hypothesis. Despite only limited support for the overarching hypothesis, important findings did arise from these investigations. In this final chapter, the key findings of the four studies are summarized and interpreted against the two broad aims of the thesis; the implications for these findings for both psychological theory and clinical practice are then discussed. Finally, the strengths and limitations of this thesis, and directions for future research, are outlined.

10.2 Aim one: Summarise the current state of knowledge regarding the male:female prevalence ratios of externalising disorders during middle childhood in non-referred children.

The results of Study 1 demonstrated that the prevalence of both ADHD and CD is higher in boys than girls during middle childhood in non-referred samples (between 2.2-3.6:1; Erskine et al., 2013; Polanczyk et al., 2007; Willcutt, 2012; Wittchen et al., 2011; and 2.4-3.0:1; Erskine et al.; Wittchen et al.; respectively). Study 2 provided a meta-analysed male:female prevalence ratio of 1.59:1 for ODD during middle childhood in non-referred samples. The results of Study 2 also demonstrated that the prevalence of ODD is significantly higher in boys than
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Several notable findings arose from Studies 1 and 2 that warrant further discussion. First, the male preponderance in prevalence appears to be less pronounced for ODD than it does for ADHD and CD. One potential reason for this, as offered by Demmer, Hooley, Sheen, McGillivray and Lum (2017), may be the nature of the symptom criteria across these disorders. ODD symptoms appear to be less action- and activity-based than the symptoms of ADHD and CD. Instead, ODD symptoms appear to be mood- and attitudinal-based (APA, 2013). Therefore, ODD symptoms may be symptomatically less externalizing and more internalizing in nature than symptoms of ADHD and CD, and the attenuation of male dominance in ODD prevalence may reflect the hypothesis that girls are socialised towards internalising presentations and boys are socialised towards externalizing presentations (Keenan & Shaw, 1997).

Second, Study 2 found that significant differences in the prevalence of ODD occurred in Western, but not non-Western, cultures. Given that the prevalence of ODD has not been found to vary across cultures (Canino, Polanczyk, Bauemeister, Rohde & Frick, 2010), the lower sex ratio in prevalence found in non-Western cultures suggests that ODD is either relatively less prevalent in boys, or more prevalent in girls, from non-Western cultures when compared to Western cultures. One potential reason for this, as offered by Demmer et al. (2017), is that non-Western cultures, where gender roles are generally more traditional (Arnold, Choe & Roy, 1998; UNICEF, 2006), may have a lower threshold for ODD behaviours in girls compared to Western cultures. Therefore, girls in non-Western cultures may be rated as more symptomatic, or be more likely to be referred
and/or diagnosed, than girls from Western cultures who display identical symptoms. This idea requires future investigation in cross-cultural research.

10.3 Aim two: Explore how (i) parenting, and (ii) ADHD diagnosis, might differ between boys and girls and thus influence the gender imbalance in ADHD prevalence

10.3.1 Parenting

Chapter 6 of this thesis suggested two ways in which the association between parenting and EDs might differ based on child gender to influence the male preponderance in ED prevalence. First, it was suggested that the association between less-than-optimal parenting and child EDs that has been found in previous research (e.g., Lifford, Harold & Thapar, 2008 Keown, 2012) might be significant for sons but not daughters. Second, it was suggested that even if the associations between less-than-optimal parenting and child ED symptoms hold for sons and daughters, sons might receive higher levels of less-than-optimal parenting than daughters, putting sons at greater risk of developing an ED. Both of these possibilities could help to explain the male preponderance in ED prevalence. Study 3 investigated these ideas by examining the potential prospective relationships between mothers’ and fathers’ Angry, Warm and Consistent Parenting dimensions and child ADHD symptoms diagnosis, and whether these relationships differed based on child gender. Results demonstrated that overall, mothers’ Angry Parenting and ADHD symptoms shared a positive, prospective, and bi-directional relationship between child ages of 4- to 7-years. The same relationship was found for fathers’ Angry Parenting practices and ADHD symptoms, however this relationship extended over a longer period, from child ages 4- to 9-years. Higher levels of child ADHD symptoms continued to
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predict higher levels of mothers' and fathers' angry parenting throughout all waves even after the bi-directional effects had ceased. Higher ADHD symptoms when children were 4- to 5-years of age also tended to predict less-than-optimal parenting for both mothers’ and fathers’ when children were aged 6- to 7-years (e.g., lower warm and consistent parenting, and higher angry parenting), however these patterns were not identical for mothers and fathers. The second latent growth curve model supported these results, finding that increases in child ADHD symptoms over time significantly predicted increases in angry parenting of both mothers and fathers. the results of the logistic regression also demonstrated that angry parenting directed towards children at ages 4- to 5-years significantly predicted an ADHD diagnosis when children were 8- to 9-years of age. although there were some differences between the findings of study 3 and previous predictive research in this area (e.g., keown, 2012; lifford et al. 2008), this may be due to methodological differences as discussed in chapter 7.

however, (i) the results of study 3, and (ii) the paucity of research in this area demonstrate that further work is needed in this area. these findings also point to the importance of including both mothers and fathers in investigations and statistical models.

these significant relationships between angry, warm and consistent parenting and child ADHD did not differ based on the gender of the child. therefore, the results of study 3 did not support the suggestion that the associations between parenting and child ADHD might be significant for sons but not daughters, and instead, suggested that the ADHD symptoms expressed by sons and daughters are equally affected by, and influence, the angry, warm and consistent parenting of mothers and fathers. therefore, it may be that existing
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Empirical knowledge regarding the associations between parenting and ADHD, which has tended to focus on male samples, might also be applicable to females. However, it will be important for future investigations to consider other parenting constructs and child gender differences to continuing advancing this area of knowledge.

The lack of child gender differences in the associations between parenting and ADHD in Study 3 are not consistent with the findings of Tung, Li and Lee (2012) who reported positive relationships between parents’ inconsistent discipline and children’s antisocial and rule-breaking behaviours in sons but not daughters. There may be several reasons for the differences between the Tung et al. results and the findings of Study 3. First, it is probably that there are inherently different relationships between parenting and CD (tested by Tung et al.) and parenting and ADHD (tested in Study 3), despite both being EDs. Second, Tung et al. examined cross-sectional relationships while Study 3 focused on prospective relationships. It might be that differences exist between the cross-sectional and longitudinal relationships. For example, given that the levels of both less-than-optimal parenting and ADHD symptoms are higher for boys, the relationships between these might develop sooner for boys than they do for girls. Conversely, as girls receive lower levels of less-than-optimal parenting and have lower ADHD symptoms than boys, it may take longer for these influential relationships to appear, and may require longitudinal research in order to expose these associations.

The results of Study 3 did provide some support for the suggestion that sons might receive higher levels of less-than-optimal parenting than daughters, thereby increasing their risk of ADHD and influencing the male preponderance in ADHD
prevalence. Across all waves, sons compared to daughters, received higher levels
of Angry Parenting from both mothers and fathers, and also had higher levels of
ADHD symptoms. It may be that across earlier waves, where Angry Parenting
predicted proceeding child ADHD symptoms, the relatively higher level of Angry
Parenting received by sons contributed to their relatively higher ADHD symptoms
when compared to the lower levels of less-than-optimal parenting and lower
ADHD symptoms in girls. This would help to account for the male preponderance
in ADHD prevalence, particularly if the strength of the symptoms influences the
likelihood of a referral and subsequent diagnosis. This result could be argued to
support the socialisation hypothesis (Keenan & Shaw, 1997) and suggests that
parents may be contributing to the gender imbalance in ED prevalence rates by
socializing their boys towards, and their daughters away from, ED-related
behaviours based on certain parenting behaviours, but further research is needed.

10.3.2 Diagnosis of externalising disorders

Using a mixed factorial design, Study 4 examined whether child gender
influenced the diagnostic decisions of clinicians when assessing children for
ADHD. The primary hypothesis of Study 4, that there would be a higher
proportion of ADHD diagnoses in vignettes involving boys than girls, was not
supported. Whether tested cross-sectionally or longitudinally, child gender did not
appear to influence the diagnostic decisions of participants, with clinicians no
more likely to provide a diagnosis or working hypothesis (WH) of ADHD for
vignettes involving boys compared to vignettes involving girls. Further, there was
no significant difference in clinicians’ confidence when provided a diagnosis or
WH of ADHD for vignettes involving boys compared to vignettes involving girls.
The finding that child gender did not influence the diagnostic decisions of clinicians is inconsistent with the results of Bruchmüller et al. (2012) and suggestions by Sciutto and Eisenberg (2007). It is possible that child gender is not an influential factor in diagnostic decision-making, and that the result of Study 4 appropriately represent the decision-making processes in Australian clinicians, and that cultural differences between the Australian and German participants in the Bruchmüller et al. may exist that influence the use of gender in diagnostic decision-making. However, the clear ceiling effects for ADHD diagnosis across all vignettes and for both genders limited the variance available to appropriately investigate the hypothesis of Study 4. Further, it is possible that the low sample size precluded a thorough investigation of gender effects. Future research is needed replicating the methodology of Study 4 in a larger sample, and perhaps across cultures.

The results of Study 4 demonstrated a propensity for clinicians to provide a diagnosis or WH of ADHD in vignettes where full diagnostic criteria were not met. This supports previous suggestions (e.g., Desgranges, Desgranges & Karsky, 1995; Goldman, Genel, Bezman, & Slanetz, 1998; Lilienfeld & Arkowitz, 2013; Partridge, Lucke, & Hall, 2014; Sciutto & Eisenberg, 2007; Shaw, Wagner, Eastwood & Mitchell, 2003) that there is a tendency for the misdiagnosis of ADHD. Taken together with our finding that gender did not appear to influence diagnostic decisions, ADHD misdiagnosis appears to occur for both boys and girls. Interestingly, the study found that the reverse occurred for major depressive disorder (MDD), with clinicians often failing to provide a diagnosis or WH of MDD when full diagnostic criteria were met.
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The comparison between rates of false positives for ADHD and false negatives for MDD supports the suggestion by Sciutto and Eisenberg (2007) that clinicians may misattribute the symptoms that occur due to another disorder (in this case, MDD) to ADHD. The mechanism by which this may occur is unclear. It is possible that as MDD only impacts approximately 1% of children (Lawrence et al., 2015), the presence of externalizing symptoms leads clinicians to (i) automatically consider ADHD and seek to confirm this diagnosis rather than disprove it, (ii) attribute symptoms of another disorder to ADHD, and/or (iii) minimize symptoms that potentially relate to another disorder and focus on ADHD-specific symptoms. This may work to increase the diagnoses of higher prevalence childhood disorders (e.g., ADHD) and limit diagnoses of lower prevalence childhood disorders (e.g., MDD).

10.4 Implications of this thesis for existing theory

The results of this thesis provide some support for the socialisation hypothesis outlined by Keenan and Shaw (1997). Keenan and Shaw posited that while girls and boys may experience the same underlying vulnerabilities to psychological difficulties and expression of problem behaviours during early childhood, they are socialised to exhibit their difficulties in different ways as they develop. Specifically, socialisation agents such as parents, teachers and peers encourage girls to express their difficulties as internalizing problems, which are more accepted and normative of the traditional female gender role than are externalising behaviours. This idea is supported by research showing there are few gender differences in externalizing behaviours during early childhood, with gender differences becoming most notable in middle childhood, following the potential
influence of socialization (Hay, 2007; Keenan & Shaw, 1997; Loeber & Hay, 1997).

The findings of Study 2, that the male:female prevalence ratio of ODD (1.59:1) appears markedly lower than those of ADHD and CD (2.2-3.1:1 and 2.4-3:1 respectively; Erskine et al. 2013; Willcutt, 2012; Wittchen et al. 2011), is consistent with the socialization hypothesis. That is, ODD may be less ‘externalizing’ than ADHD and CD, and therefore girls may not be socialized away from ODD-related behaviours as they are from ADHD- and CD-related behaviours. An examination and comparison of the diagnostic criteria of these disorders provides some support for this argument, with ODD diagnostic criteria potentially reflecting attitudinal- and/or mood-orientated symptoms (e.g., issues with temper, disobedience against authority, vindictiveness), while ADHD and CD diagnostic criteria (with the exclusion of the ADHD inattentive subtype) reflecting action- and/or activity-orientated symptoms (e.g., physical fighting and cruelty, hyperactive behaviours such as fidgeting and difficulties remaining seated). This re-conceptualization of the externalising nature of ODD deviates from general understandings of ODD (e.g., Burke, Loeber & Birmaher, 2002), and some contention may exist regarding the nature of the mood component of ODD; for example, whether mood is an internalised (i.e., inwardly directed) or externalised (i.e., outwardly directed) symptom of the disorder. However, based on the findings of Study 2, consideration that ODD may in fact be less externalising than other EDs warrants consideration.

The gender difference in the prevalence of ADHD subtypes is also consistent with this suggestion showing a greater gender imbalance in the hyperactive/impulsive and combined subtypes (the more externalising subtypes)
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than in the inattentive subtype (the less externalising subtype; Willcutt, 2012). This explanation appears to fit within the claims of Keenan and Shaw (1997): if (i) girls are socialized to manifest their difficulties internally and not externally, and (ii) ODD and ADHD inattentive subtype are less externalizing and more internalizing in symptomology than other EDs, then it would be expected that there would be an attenuation of the male dominance in the prevalence of ODD and ADHD inattentive subtype compared to ADHD combined and hyperactive/impulsive subtypes and CD.

Study 3 also provides some support for the socialisation hypothesis (Keenan & Shaw, 1997). Daughters received lower levels of certain parenting dimensions that were related to ADHD symptoms, and had lower ADHD symptoms than sons, which is consistent with the idea that parents may socialise boys towards ADHD, and girls away from ADHD, by exposing their sons and daughters to differing levels of less-than-optimal parenting. It appears that boys may be at a greater risk of ED development than girls by receiving higher levels of Angry Parenting as measured in Study 3. These results suggest the socialisation hypothesis may be a useful framework through which to understand the relationship between parenting and EDs. It may therefore be beneficial for future investigations into the socialisation of children towards, or away from, EDs on the basis of gender to adopt this framework.

10.5 Implications of this thesis for clinical practice

Several implications for the assessment, diagnosis and treatment of EDs arose from the results of this thesis.
10.5.1 Culture and child gender in the assessment and diagnosis of externalising disorders

The results of Study 2, that significant gender differences exist in the prevalence of ODD in Western, but not non-Western, cultures suggests that it is important for clinicians to consider the role of societal expectations and standards in the assessment and diagnosis of ODD, and potentially other EDs as well. As the overall prevalence of ODD is not found to vary across cultures (Canino, et al., 2010), it may be that in non-Western cultures, where gender roles and expectations are more traditional (e.g., externalizing and disruptive behaviours might be less acceptable in girls than boys; Arnold et al., 1998; UNICEF, 2006), problem behaviours displayed by girls might have a lower threshold of concern compared to Western cultures, leading girls to be more likely referred with subclinical symptoms compared to boys.

It appears important for clinicians to be mindful of cultural considerations (both their own and of the referred child/family) in their assessment of ODD as differences in gender roles and expectations across cultures may impact the identification of ODD (and possibly other EDs). For example, the thresholds at which ODD-related behaviours are considered pathological, and at which a diagnosis of ODD might be considered or applied, may depend on the culture in which the behaviours are present, because the context in which externalising behaviour occurs is inescapably a social one. Thus, the decision as to whether or not a child’s behaviour is disruptive is a social judgment, and it is likely that different societies and cultures make different judgments (Canino & Alegria, 2008), including what they regard as appropriate behaviour for boys and appropriate behaviour for girls. It is important that clinicians are aware of how (i)
their own cultural biases in relation to gender roles and gender-based expectations may influence their clinical decisions involving assessment, diagnosis and treatment of EDs, and (ii) the cultural biases of parents and other referral agencies may influence their interpretation of the behavior of children displayed by both boys and girls who are subsequently presented for evaluation.

**10.5.2 Diagnosis of ADHD**

The propensity of clinicians in Study 4 to provide a diagnosis or WH of ADHD regardless of presentation supports previous suggestions, both in research and in public opinion, that ADHD may be misdiagnosed (e.g., Desgranges et al., 1995; Goldman et al., 1998; Lilienfeld & Arkowitz, 2013; Partridge et al., 2014; Sciutto & Eisenberg, 2007; Shaw, Wagner, Eastwood & Mitchell, 2003). However, results suggested that child gender was not influencing this misdiagnosis trend; leaving unanswered the question as to what factor/s influence/s are at play here. Several factors that may instead influence clinicians’ diagnostic decisions are worthy of future consideration. First, some clinicians may fail to follow best-practice guidelines (such as the use of diagnostic criteria from diagnostic manuals) in the assessment and diagnosis of childhood psychological disorders (Handler & DuPaul, 2005; Sciutto & Eisenberg, 2007), and instead may rely exclusively on clinical judgment. Diagnostic manuals and best-practice guidelines for assessment are critical to ensure appropriate diagnoses, where appropriate, are reached.

Second, it may be that clinicians are not engaging sufficiently in the process of differential diagnosis. Clinicians may be seeking to confirm the presence of specific disorders (in the case of Study 4, ADHD), and not considering, or seeking to disconfirm, the presence of other disorders. Study 4 demonstrated that the
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presence of a disorder that can share similar features to ADHD (i.e., MDD) might lead clinicians to suspect and/or diagnose ADHD (a possibility suggested by others; e.g., Sciutto & Eisenberg, 2007) and foreclose on this decision before completing the differential diagnostic process. Reasons for foreclosure and conditions under which foreclosure occurs requires further investigation because it is possible that foreclosure affects the veracity of other decisions/diagnoses made by clinicians. Foreclosure resulting in misdiagnosis or missed diagnosis might have significant impacts on children who may be subsequently (i) exposed to inappropriate treatments and the potential side effects of these treatments (in the case of ADHD, potential psychopharmacological treatments and their side effects), (ii) deprived of appropriate treatments to negate the detrimental outcomes associated with untreated conditions, and/or (iii) deprived of financial assistance and educational support that is often attached to diagnoses of childhood disorders. It is critical for clinicians to engage in a process of differential diagnosis to ensure that alternative hypotheses and disorders are considered for all clients.

10.5.3 Treatment of ADHD

Study 3 provided evidence to support the importance of social-level factors in the development and/or maintenance of ADHD; these findings encourage consideration of the potential psychosocial treatments for this disorder. Although frontline treatment of ADHD is generally stimulant medication, psychosocial interventions play an important role in the treatment process for several reasons. First, a proportion of parents of children with ADHD decide against medication use for their child’s difficulties, often due to the side effects of such medication which can include decreased appetite and insomnia (Schachter, Pham, King, Langford & Moher, 2001). Second, there is a significant issue with medication
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non-adherence and/or discontinuation in ADHD (estimated to be between 13.2% and 64%; Adler & Nierenberg, 2010), demonstrating that a large proportion of children treated pharmacologically requires alternate interventions. Third, it has been suggested that combined medication and psychosocial treatments may enhance positive outcomes and produce longer-term improvement across several domains (Klein, Abikoff, Hechtman & Weiss, 2004), as medication has little impact on the factors that psychosocial interventions often target, such as parenting.

The results of Study 3 suggest some important considerations for the psychosocial treatment of ADHD, and perhaps EDs more generally. First, the results suggest that increasing Warm and Consistent Parenting, and decreasing Angry Parenting, during the years just prior to middle childhood, may lead to lower child ADHD symptoms as children enter middle childhood. These specific parenting dimensions could be considered in the development of future psychosocial treatments for ADHD that intervene in parenting behaviours (e.g., parent training).

Second, the results of Study 3 highlight the important association between fathers’ parenting and child ADHD that has often been ignored in research into the psychosocial treatment of ADHD (Fabiano, 2007). The results of Study 3 demonstrated that maternal parenting and paternal parenting shared different prospective relationships with child ED symptoms. This finding supports previous claims (e.g., Braza, et al., 2015; Keown, 2012; Lamb, 2004; Lifford et al., 2008) that child developmental outcomes differ depending on whether maternal or paternal influences are considered, and therefore suggest including fathers in psychosocial interventions for ADHD (and possibly other EDs) may provide
specific beneficial outcomes for both children and their parents above and beyond just the inclusion of mothers. As child gender did not moderate the relationship between Angry, Warm and/or Consistent Parenting and child ADHD, it is possible that interventions focused on altering parenting practices will be effective for both boys and girls.

10.6 Strengths of this thesis

This thesis attempted to empirically investigate how social-level factors associated with the development and diagnosis of EDs might impact girls and boys differently to influence the gender differences in ED prevalence rates. Previous researchers have posited that EDs are poorly understood in girls (e.g., Gaub & Carlson, 1997; Gershon 2002), and this thesis has made attempts to extend current knowledge in that area. The thesis summarized what is known regarding gender differences in the prevalence of ADHD and CD during middle childhood in non-referred samples, and provided the first meta-analysed male:female prevalence ratio of ODD during middle childhood in non-referred samples. This has brought together the most current and wide findings regarding the gender imbalance in ED prevalence rates. Further, the thesis is the first to explore how the bidirectional, prospective relationships between three key parenting dimensions and ADHD might differ between boys and girls, given that similar previous research has not assessed how these relationships might differ based on child gender (e.g., Keown, 2012; Lifford, Harold & Thapar, 2008). Given the close association between child gender and both parenting and EDs, this has been a notable omission of previous research in this area. From this, the current thesis has been able to suggest that the male preponderance in ADHD prevalence may be related to boys receiving higher levels of certain less-than-
optimal parenting practices that are related to ADHD development, and perhaps may not be due to the overall relationships between parenting and ADHD differing based on child gender.

Finally, the research reported in this thesis is one of the first to explore how clinicians might be making errors when assessing and diagnosing children with problem behaviours. Although contrary to the hypothesis, child gender did not arise as a determinant of diagnosis, important findings that arose were the misdiagnose of ADHD in the form of false positives, and the misdiagnosis of MDD in the form of false negatives. Although these findings require further empirical investigation to determine the mechanism that is driving these misdiagnoses, as is highlighted in the current discussion, they should stand as a caution and prompt to clinicians regarding their diagnostic decision-making processes.

10.7 Limitations of this thesis

Despite the strengths of this thesis, and the novel investigations and findings herein, several limitations that impacted its ability to test for the influence of gender on the development and diagnosis of EDs should be considered when interpreting the findings. This thesis explored only two social-level factors: parenting and diagnosis. There are numerous other socialization agents and factors that may be of influence in the development of EDs, for example, peers and teachers. It might be that parents are not in fact as important in the socialisation of daughters and sons as has been suggested in previous research, and thus they may be of only minimal influence in the male:female prevalence ratios of EDs. However, further research in this area is needed before such a conclusion can be drawn. Further, as ADHD-I does contain predominantly
internalising features in its presentation (APA, 2013), the findings of this thesis may not generalise to ADHD-I as they might to ADHD-HI and ADHD-C. Where possible the potential internalising nature of ADHD-I has been acknowledged throughout this thesis in terms of how it might differ from the more externalising presentations of ADHD. Future research specifically examining child gender differences in the development of ADHD-I may be beneficial, and might potentially find similar results to those found for ODD in Study 2 of this thesis given ADHD-I’s internalising nature.

Third, Studies 3 and 4 were limited to testing the relationships between parenting, diagnosis and ADHD symptoms only, due to the size constraints of this thesis. Therefore, although ADHD shares many similarities to CD and ODD, the findings of Studies 3 and 4 cannot be generalized to these other EDs. It is possible, and likely, that Warm, Angry and Consistent Parenting share different relationships with both CD and ODD than they do with ADHD and this needs to be explored. However, given ADHD sees greater gender differences in prevalence compared to CD and ODD, the use of ADHD as an example of an ED seemed to provide a good opportunity to test child gender influences in the development of the disorder.

It was originally planned to include both mothers’ and fathers’ responses on the SDQ in Study 3. Unfortunately, due to considerable levels of missing data for fathers’ responses, only mother responses were used to determine the level of ADHD symptomology. As there is often little consistency between the SDQ scores as rated by mothers, fathers and/or teachers (Stokes, Mellow, Yeow & Hapidzal, 2014), different results may have been found if different respondents were used. Further to this point, other measures of ADHD symptomology may
have yielded different results. Although the SDQ is a valid and sensitive screening tool for ADHD symptoms (Goodman, Ford, Simmons, Gatward & Meltzer, 2000; Stone, Otten, Engels, Vermulst & Janssens, 2010), a measure such as the Child Behaviour Checklist (CBCL), which assess a wider range of ADHD-related behaviours and outcomes, may have provided a more valid picture of child ADHD symptomology. However, there are some suggestions that the SDQ is superior to the CBCL in detecting ADHD-related symptomology (Goodman & Scott, 1999). In addition, it may be that the parenting and/or ADHD measures used in Study 3 were inadequate. As was the case with the variables available for extraction from the LSAC dataset, large longitudinal studies often need to be expedient in their measures and thus select small-item screening scales which are perhaps less valid and reliable than larger, more comprehensive clinical measures. Further, only a limited number of parenting dimensions were available from the dataset for use. These dimensions represent only a narrow selection of a wide spectrum of parenting dimensions, practices and/or styles that might be influenced by child gender, or related to ED presentation.

The sample size of Study 4 was insufficient to perform sophisticated statistical analyses on the data, resulting in only descriptive statistics and trends in the data being reported. Due to this limitation, it may be appropriate to draw only tentative inferences from Study 4, particularly given its results do not support previous findings in a larger sample (Bruchmüller, Margraf & Schneider, 2012). Several other limitations in the design of Study 4 also need to be considered. First, the use of vignettes provided only limited information to participants and did not reflect the wealth of assessment information that is often available to clinicians when making diagnostic decisions. The inclusion of further information such as
measures of the child’s social functioning, intelligence and academic performance may have changed participant responses. Therefore the ecological validity of the study is a concern and it is possible that the high rate of false positive diagnoses of ADHD observed in Study 4 is an overestimation of that occurring in clinical practice. However, concerns about the ecological validity of the vignettes were partially addressed by having a team of clinical psychologists with a specialisation in the area of child and adolescent psychology review the vignettes, with modifications made on the basis of their expert feedback. Finally, as this thesis investigated middle childhood specifically, it may be inappropriate to generalize the findings to other periods of child development (e.g., early childhood and/or adolescence).

10.8 Directions for future research

Research into potential sex and gender differences in the development and diagnosis of EDs requires further investigation if we are to understand what is influencing the male preponderance in ED prevalence. As suggested in the discussion of Study 3, an important next step will be to investigate if child gender moderates the relationship between parenting dimensions other than Warm, Angry and Consistent Parenting. These three dimensions represent only a small collection of parenting dimensions, styles and practices that have been associated with child EDs. For example, child gender differences might exist in the associations that have been found between child EDs and parental controlling behaviours (Rogers, Wiener, Marton & Tannock, 2009), parental overprotection (Chang, Chiu, Wu & Gau, 2013), and parental responsiveness (Landau, Amiel-Laviad, Berger, Atzaba-Poria & Auerbach, 2009). These warrant further research.
Further to this, it might be that girls and boys are differentially sensitive to parenting behaviours based on biological, neurological or developmental gender differences. For example, similar parenting behaviours may lead to different reactions in boys and girls, and these gender-based sensitivities may increase the risk of the development of EDs in boys, or decrease the risk of ED development in girls. This suggestion too warrants further investigation. It will also be important for future research to consider how the relationships between EDs and other socialisation agents, such as teachers and/or peers, might differ based on child gender.

The findings of Study 4, that clinicians demonstrated a propensity to diagnose ADHD regardless of child gender, necessitates further investigations into other factors influencing diagnostic inaccuracies. In their summary of evidence regarding the over-diagnosis of ADHD, Sciutto and Eisenberg (2007) highlight potentially fruitful avenues of investigation into the causes of ADHD misdiagnosis that require future investigation. These include (i) inadequate considerations of differential diagnoses involving disorders commonly comorbid with ADHD (i.e., difficulties with attention due to mood or anxiety disorders rather than ADHD), (ii) diagnostic inaccuracy (e.g., clinicians failing to follow best-practice guidelines in their assessment and diagnosis of childhood disorders), (iii) changes to diagnostic criteria (in that more recent versions of diagnostic manuals may increase the chances of an ADHD diagnosis), (iv) ignoring potential sub-norm groups (e.g., different cultures) and (v) barriers to identification and treatment (in that a large percentage of children with mental health difficulties do not seek attention for these issues). Improving empirical understanding about
factors influencing ADHD misdiagnosis will be crucial to inform clinical practice and ensure childhood difficulties are being appropriately recognized and treated.

It will be important for future research to also explore CD and ODD in regards to how the development and diagnosis of these disorders might differ based on child gender. Although ADHD, CD and ODD all fall under the classification of EDs, it is inappropriate to generalize the findings of studies three and four across EDs. It is hoped the methodology of Studies 3 and 4, and any possible modifications to these methodologies based on their limitations, can act as a framework for similar investigations where CD and ODD may be the disorders of interest rather than ADHD. Given ADHD has one of the highest heritability aetiologies of any psychiatric disorder, it may be that environmental factors are less influential in the development of ADHD than they are for CD and/or ODD, and child gender may have a greater role in the development of the latter two disorders, supporting the importance of further investigations in this area. For this reason, research into CD and ODD is needed, and may be particularly fruitful. It will be important to consider the role of Disruptive Mood Dysregulation Disorder (DMDD), a childhood disorder introduced in the DSM 5 (AP, 2013), in these investigation given its diagnostic similarities to ODD. Although the research base on DMDD remains small, there is potential for it to be classified as an ED, warranting its inclusion in future ED investigations.

Although this thesis has specifically focused on social-level factors, it is important to acknowledge that it is estimated that approximately half of the variance associated with the development of EDs can be accounted for by biological factors (Derks, Dolan, Hudziak, Neale & Boomsma, 2007). Therefore biological explanations for the gender differences in the prevalence of EDs also
require future attention. For example, differences between girls and boys may exist in the DRD4, DAT1, DRD5, 5-HTT and HTR1B genes that are important in the development of ADHD (Bobb, Castellanos, Addington & Rapoport, 2006; Farone & Doyle, 2000; Hicks, Krueger, Iacono, McGue & Patrick, 2004; McCracken, et al., 2000; Slutske, et al., 1997; Thapar, Cooper, Eyre & Langley, 2013; Thapar, Hervas & McGuffin, 1995), however sex differences are not often examined in relation to these biological aetiologies. Exploring both biological and social factors may assist in also understanding any epigenetic effects that may be occurring.

10.9 Conclusion

This thesis synthesized results that demonstrate a male preponderance in the prevalence of EDs. This finding suggests that child gender is related to the occurrence of these disorders. Until now, few investigations have explored how factors related to the development and/or diagnosis of EDs might differ between boys and girls to influence the gender imbalance in prevalence rates. Overall, the findings of the current thesis provided only minimal support for the suggestion that boys may be socialised towards ED-related behaviours, and girls may be socialised away from ED-related behaviours, by receiving differing levels of certain less-than-optimal parenting behaviours.

It is important, however, to recognize that the factors examined in this thesis (i.e., parenting and ED diagnosis) are just a small collection of the broad range of social-level factors that may influence EDs. It is now important for future research to examine other social-level factors, such other parenting dimensions, practices and styles, as well as other socialisation agents such as teachers and peers, to explore how these might be differentially associated with EDs based on child
gender. Further investigation is also needed into the factors that may be influencing the misdiagnosis of ADHD as demonstrated in the results of this thesis. To further develop empirical understanding of EDs, and to ensure the appropriate ED diagnosis and treatment of EDs for both genders, it is imperative that work continues to progress understanding of the factors that may influence the gender imbalance in ED prevalence.
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Appendices
Sex Differences in the Prevalence of Oppositional Defiant Disorder During Middle Childhood: a Meta-Analysis

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Abstract. This review provides a meta-analysis of the gender prevalence ratio of oppositional defiant disorder (ODD) during middle childhood in non-referred children. It also analyses sex differences in prevalence across cultures and over time. A systematic search for studies via the following sources was conducted: PsycINFO, Web of Knowledge, Medline Complete, Scopus, EMBASE, InfoTR, Psychological and Behavioural Sciences Collection, Cochrane Library, PubMed and ProQuest Health. The studies presented in two previous systematic reviews were also added to the search results. Inclusion/exclusion criteria were then applied and final studies were appraised for their methodological quality. Nineteen independent effect sizes met full inclusion criteria (aggregated sample N = 44,107). Overall, the prevalence of ODD was significantly higher in boys than girls (RD = 1.59, 95% CI [1.30, 1.88], p < 0.001), with the male-to-female prevalence ratio found to be 1.5:1. Sex differences in prevalence were significant in Western (RR = 1.80, 95% CI [1.55, 2.09], p < 0.001) but not non-Western cultures (RD = 1.08, 95% CI [0.76–1.53], p = 0.05). Sex differences in prevalence were significant in studies published prior to and post the year 2000 (RD = 1.57, 95% CI [1.22, 2.02], p < 0.001, RR = 1.84, 95% CI [1.35, 2.00], p < 0.001), and were consistent between these two periods (Q, I(2) = 0.38, p = 0.5). The sex differences in ODD prevalence are discussed within the context of (i) predominant theories of sex differences in externalizing behaviors, and (ii) departures from the sex differences pattern found for other disruptive behavioral disorders.

Keywords. Childhood - Oppositional defiant disorder - Psychopathology - Development - Gender

Oppositional Defiant Disorder (ODD) is characterized by a persistent pattern of angry/irritable mood, disobedient and hostile behavior towards authority figures, and/or vindictiveness (American Psychiatric Association [APA], 2013), with population prevalence estimates ranging from 1.4 % (Asher et al. 2001) to 12.3 % (Cohen et al. 1993). Symptoms generally begin during the preschool years, with diagnosis typically occurring during middle childhood (i.e., 6- to 13-years-of-age; Kessler et al. 2003). ODD is associated with a range of detrimental outcomes including emotional and peer problems (Munkvold et al. 2011), family and social dysfunction (Greene et al. 2002), psychiatric comorbidity (Loeb et al. 2003), high mental health service utilization (Cohen et al. 1993), and is seen as a predictor in the development of conduct disorder (CD) and some personality disorders (Burke et al. 2002; Hoiles et al. 1991; Loeb et al. 1995; Maughan et al. 2004).

Due to commonalities between ODD, Attention-Deficit/ Hyperactivity Disorder (ADHD) and CD in terms of symptoms, age of onset, comorbidities, psychosocial treatment, and mental health service utilization (Bedrosian et al. 2005; Burke et al. 2002; Greene et al. 2002; Kendler et al. 2002; Merikangas et al. 2011; Reid et al. 2004), these three disorders...
are commonly grouped under the umbrella term of disruptive behavioural disorders (DBDs). A further similarity between DBDs is the marked sex differences in their prevalence rates, with all three disorders more common in boys than girls. According to recent meta-analyses, the male:female prevalence ratio for ADHD is between 2.2:1 and 3:1 (Erskine et al. 2013; Wilke et al. 2012; Wittchen et al. 2011), and between 2.4:1 and 3:1 for CD (Erskine et al. 2013; Wittchen et al. 2011) during middle childhood and adolescence. Although it is suggested ODD follows similar trends (Boylan et al. 2007; Canino et al. 2010), a meta-analysis to quantify this is yet to be conducted. This is an important investigation for several reasons. First, much of the published literature reports no statistically significant sex difference in ODD prevalence (e.g., Angold et al. 2002; Cohen et al. 1993; Leung et al. 2008; López-Villalobos et al. 2014; Mishra et al. 2014; Niemczyk et al. 2014; Park et al. 2014; Simonoff et al. 1997), thus clarity on whether one exists is needed. Second, confirming the male:female prevalence ratio of ODD invites speculation about, and offers potential avenues for, research into the aetiology and maintenance of the disorder (Aleman et al. 2003; Zahn-Waxler et al. 2008). The higher occurrence of ODD in males would imply that there is something about being male that increases the risk of ODD developing/being maintained, and/or something about being female that protects against this risk.

Theories About Sex Differences

Various theories accounting for sex differences in the prevalence of ODD, other DBDs, and externalizing problems in general, have been offered. These suggest biological factors (Caspi et al. 2002; Eme 2007; Lahey et al. 2011), pathways in the development of DBDs (Crick and Zahn-Waxler 2003; Moffitt 1993; Moffitt and Caspi 2001), and differences in manifestations of externalizing problems (Crick and Zahn-Waxler 2003) may be influential. Biologically, it is estimated that approximately half the variance associated with the development of externalizing problems can be accounted for by biological factors (Derks et al. 2007). For example, genetic research demonstrates the monoamine oxidase A (MAOA) and dopamine transporter (DAT1) genes are linked to conduct behaviours (Caspi et al. 2002; Lahey et al. 2011), and that these may interact with environmental factors (such as parenting) to increase the difficulties with self-control that underlie delinquency behaviours (Watts and McNulty 2014). Eme (2007) also discusses further potential leads in this area that warrant investigation; particularly why girls with congenital adrenal hyperplasia (CAH), a fetal disorder that leads girls to be exposed to male levels of androgens in utero, tend to have high rates of behavioural problems. Despite this, a common limitation of biological studies in this area is the failure to compare male and female participants, inadvertently hindering the exploration of a biological basis of sex differences in prevalence rates. Thus, how biological pathways in DBD development differ for boys and girls remains unclear (Burke et al. 2002; Derks et al. 2007; Moffitt et al. 1998).

Boys and girls follow different trajectories in the development of antisocial behaviours. Boys regularly follow either a child-onset pathway (the development of antisocial behaviours during early or middle childhood), or an adolescent-onset pathway (the development of antisocial behaviours during adolescence; Crick and Zahn-Waxler 2003; Moffitt 1993; Silverthorn and Frick 1999). In contrast, the child-onset pathway in girls is rare (10:1 in favour of boys; Eme 2007; Moffitt and Caspi 2001), with most girls who develop antisocial behaviours doing so during adolescence (1.5:1 in favour of boys; Moffitt and Caspi 2001). This likely impacts the male:female prevalence ratios of DBDs during early and middle childhood. It is expected that girls might be protected against child-onset due to various factors including their earlier physical maturation, and better-developed language, social and emotional skills compared to boys (Crick and Zahn-Waxler 2003; Keenan and Shaw 1997). For both genders the child-onset pathway is particularly important to understand as it is associated with greater risk of difficulties and serious psychopathology through adulthood compared to the adolescent-onset pathway (Moffitt and Caspi 2001; Silverthorn and Frick 1999).

Gender differences in DBD prevalence rates are also likely impacted by boys and girls presenting with different manifestations of externalising issues during childhood, despite a similar underlying mechanisms driving both presentations (Crick and Zahn-Waxler 2003). This perspective is seen in the idea that diagnostic criteria for ODD may fail to capture the complete range of female presentations (Conner 2002; Ollan and Johnston 2005; Waschbusch and King 2006). For example, using sex-specific norms, Waschbusch and King (2006) identified a group of untreated girls who did not meet DSM criteria for an ODD diagnosis, yet had elevated ODD symptomatology and were almost as functionally impaired as girls who did meet criteria. Almost no boys were identified when applying the same method. This suggests boys are appropriately identified under existing DSM diagnostic criteria for ODD, however there may exist a group of impaired girls who are missed by the existing criteria, and thus may not be receiving the required assistance for their difficulties. This finding is not surprising given diagnostic criteria are formulated from clinical samples, which for DBDs tend to be dominated by referred males (Gershon 2002; Kann and Hanna 2000).

Sex differences in the prevalence of DBDs may also be associated with sex differences in early risk factors. As an example, less-than-optimal parenting practices have been consistently associated with the development and maintenance of DBDs (Burke et al. 2002), including low levels of parental warmth and affection (Altrudeh et al. 2007; Tripp et al. 2007),
physical aggression (Stormshak et al. 2000), and high levels of hostility (Harold et al. 2013). Less-than-optimal parenting practices appear especially predictive of oppositional and aggressive behaviours in children (Stormshak et al. 2006). As boys are more likely to receive less-than-optimal parenting than girls (de Ancos and Assato 2011; Lloyd and Devine 2006; Maloney et al. 2006), boys appear to have higher exposure to this risk factor, possibly increasing the occurrence of ODD in boys. Although parenting practices appear to be a logical causal argument for the aetiology of DBDs, it is important to acknowledge the bi-directional relationship between parenting and child behaviour (Burke et al. 2008). Thus, higher rates of ODD in boys might be both causal, and consequential, of higher rates of less-than-optimal parenting.

Other Influences on Sex Differences in Prevalence Rates

Extraneous influences that may contribute to sex differences in ODD prevalence are also an important consideration. Two of these are explored in the current meta-analysis: Culture and Time. Culture plays an important role in the presentation and understanding of mental health disorders (Canino et al. 1997). In their review of CD and ODD prevalence across cultures, Canino et al. (2010) analysed the effect of culture on CD and ODD prevalence rates, however found only methodological differences, and not geographical location, were associated with differences in prevalence rates between studies. Unfortunately, Canino and colleagues were unable to quantify the male-female prevalence rate across cultures due to the limited data available, so how the sex differences in prevalence rates may differ across cultures remains unclear. Individual studies, however, do suggest sex differences are not universal across cultures. For example, in Western samples where traditional sex roles and gender expectations are considered to be minimised (e.g., USA, Great Britain), ODD typically trends to a higher prevalence in boys than girls (Angold et al. 2002; Bird et al. 2006; Costello et al. 2003; Ford et al. 2003; Simonsen et al. 1997). However, when investigating a Chinese sample, Leung et al. (2008) found a trend for a higher prevalence of ODD in girls than boys (10.4 % versus 6.9 %), albeit this difference was not statistically significant. It may be that cultural differences in tolerance of behaviours symptomatic of ODD in boys, girls, or both, may be impacting male-female prevalence ratios. For example, the attenuation in male dominance in Leung and colleagues’ Chinese sample may relate to a greater respect for traditional gender roles in non-Western cultures (Arnold et al. 1998; UNICEF 2006). With higher expectation of feminine behaviour in females, even subclinical externalising behaviours might be perceived as aberrant in girls and be reported as such on diagnostic instruments.

Understanding if sex differences in ODD prevalence have changed over time may suggest social/environmental influences are important in the disorder’s aetiology, referral, and/or diagnosis, as these influences can change over shorter periods than biological aetiologies. The overall rate of conduct problems more than doubled during the 1970s, 1980s, and 1990s (Collishaw et al. 2004), with the increase in girls’ delinquency suggested to underlie this increase, and to have attenuated the sex difference over time (Loeber et al. 2000). These trends over time are yet to be examined for ODD.

The Current Review

The primary aim of the current review is to systematically search the published literature to provide a meta-analysed male-female prevalence ratio of ODD during middle childhood in non-referred children. Based on the sex differences in the prevalence of other DBDs, we hypothesize that the prevalence of ODD will be significantly higher in boys than girls. Two secondary aims are (i) to examine cultural differences by providing male-female prevalence ratios for Western and non-Western cultures separately, and (ii) to examine potential changes in sex differences in ODD prevalence over time.

Method

Systematic Search

A systematic search for studies via the following databases was conducted: PsycINFO, Medline Complete, Psychological and Behavioural Sciences Collection, PubMed (all via Ebscohost), Web of Knowledge, Scopus, EMBASE, InfoRMIT, Cochrane Library and ProQuest Health. The studies reported in Boylan et al. (2007) and Canino et al. (2010) were also included in the search results. Neither of these two previous reviews provided a meta-analysed male-female prevalence ratio of ODD. Boylan et al. reported only individual study sex differences, and Canino et al. did not include child sex in their analysis due to the low number of studies that provided appropriate prevalence-by-sex data for extraction. A review of the reference lists of studies included in the meta-analysis was also conducted, as was a by-hand search of Google Scholar. The following search terms were used: “rates of diagnosis” or “diagnosis rate” or “prevalence” or “incidence” or “frequency” and “oppositional defiant disorder”. No restrictions were placed on the date of publication. The authors did not conduct a deliberate search for unpublished literature. It is unlikely this has biased our results as (i) much of the published literature already reports no significant sex differences in ODD prevalence, thus it seems non-significant findings are not a common reason to
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be rejected for publication, and (ii) the primary aim of most literature reporting ODD prevalence-by-gender is to provide an overall prevalence rate (i.e., collapsed across gender) for ODD as well as various other disorders. Thus, other similar studies are unlikely to have been rejected for publication due to non-significant sex differences in ODD prevalence as this is generally reported only as a supplementary/additional analysis and finding.

Study Inclusion Criteria

Several considerations were taken into account when planning this review and study inclusion criteria. First, to develop an understanding of sex differences in disorders it is important to examine non-referred samples (i.e., general population samples; Bruckmüller et al. 2012; Rutter et al. 2003). Referred samples say little about the true prevalence of a disorder, only reflecting the number of individuals who are diagnosed and/or treated, which may in fact be a function of other factors such as disorder severity, chronicity, and/or patterns of comorbidity (Rutter et al. 2003).

Second, as mentioned, the average age of onset for ODD is during middle childhood, and as suggested by others (e.g., Boylan et al. 2007), middle childhood sees the most pronounced sex differences in the prevalence of ODD before sex differences tend to even out during adolescence. Thus, the risk and/or protective factors impacting the occurrence of ODD in boys and girls may be most predominant during this period. In light of these points, investigating middle childhood specifically should allow for the most comprehensive examination and provide the most widely generalizable and useful findings, particularly in highlighting the unequal gender distribution of ODD and its possible gender-based risk and protective factors.

Studies were included in the final analysis if they met the following criteria. First, studies needed to contain a non-referred sample (i.e., not a clinical sample) obtained via probability sampling or total sampling. Second, the sample age range needed to be within middle childhood (i.e., six to thirteen years of age). In order to increase the number of included studies, to facilitate a more robust meta-analysis, studies with an age-range outside of middle childhood were included if they reported an average age range within the middle childhood years (e.g., 5- to 15-years-of-age with an average age of 10-years). Third, ODD prevalence needed to be based on a standardized measure derived from DSM (III, III-R, IV, IV-R, 5) or ICD (9, 10) diagnostic criteria. Fourth, studies needed to report ODD point prevalence (i.e., not lifetime prevalence). Fifth, studies needed to report a male/female prevalence ratio of ODD, or numerical data that could be converted to a ratio for the purpose of meta-analysis.

Data Extraction and Meta-Analytic Procedures

Data were extracted from each study to permit calculation of a Risk Ratio (RR) and its variance of an ODD diagnosis given the sex of the participant. Specifically, female prevalence rates were extracted and converted to a percentage of the total female sample (for example, if the total sample of girls was 100, and 10 of those girls were identified as having ODD, the prevalence of ODD for girls in the study was 10%). The same procedure was then completed for the male prevalence rates. Standard errors were then calculated for each prevalence rate using the following formula: $SE = \sqrt{p(1-p)/n}$. The conversion of extracted data to a RR was undertaken using Comprehensive Meta-Analysis Software Version 2.0 (Borenstein et al. 2005). The RR was computed so that values greater than 1.0 indicate risk of ODD diagnosis was higher in males compared to females. RR values for the included effect sizes were averaged using a random-effect model (Hedges and Vevea 1998). Thus it is assumed that differences between study-level effect sizes reflect random error and systematic influences. Specifically, a random-effects model assumes differences in study-level effect sizes arise from two sources of error or variance: sampling error (within-study error), and true heterogeneity (between-study error). Between-study error arises from the influence of one or more variables that systematically increase or decrease an effect size (e.g., geographical location, time). The weighting of studies in a random-effects model takes into account both within- and between-study error variance then assigns each study a weight based on the inverse of that variance (Borenstein et al. 2007). This ensures the studies with higher numbers of participants are not simply assigned a higher weight in the meta-analysis based on their larger sample size.

Results

Search Results

A flow diagram of search results is provided in Fig. 1. The search was conducted by the first author and identified 1034 articles, with 27 additional records identified via a lateral search. A total of 607 articles remained after removing duplicates. The first author screened articles by title and/or abstract with 314 papers excluded at this stage (mostly due to studies not actually investigating ODD prevalence). The first author then examined 95 full text articles, with 74 excluded at this stage. The second author examined articles queried regarding their appropriateness for inclusion before a final decision was made. The reasons for exclusion at the full text stage are summarised in Fig. 1, and each individual reason for
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Fig. 1 Flow diagram of search results

- Records identified through database searching (n=1034)
- Additional records identified through lateral search (n=27)
- Records after duplicates removed (n=627)
- Records screened (abstracts) (n=627)
- Full-text articles assessed for eligibility (n=53)
- Studies included in assessment of bias (n=19)
- Studies included in quantitative synthesis (meta-analysis) (n=13; 19 effect sizes)
- Records excluded (n=314)
  - Full-text articles excluded, with reasons
    - Inappropriate data for extraction (e.g., unable to calculate mean and standard deviation)
    - Not an investigation of prevalence/proportion data reported
    - Not a non-referral sample or community sample
    - Not a current or recent study
    - Identical sample as earlier study
    - Only examined ODD as a clinical disorder
    - Outside age range
    - Excluded at assessment of bias level for methodological issues

Exclusion is provided in the online supplementary material. The most common reason for exclusion was inappropriate data for extraction (e.g., 38 articles did not provide separate ODD prevalence rates for boys and girls). The remaining nineteen studies were appraised for their methodological quality and scientific rigour using a scale based on the Newcastle-Ottawa Scale adapted for cross-sectional studies (Wells et al. 2000). The scale criteria appraised those components of studies that may be prone to introducing bias, and have been used to assess bias in previous meta-analyses (e.g., Herzog et al. 2013). Appraisal was conducted based on five components: sample (sampling procedure and sample size), validity of measurement tool, assessment process, number of respondents, and quality of statistical analysis (see Table 1 for specific criteria and Table 2 for scores of included studies). Each study could receive a possible score between zero and eight, with higher scores representing higher methodological quality. The first and second authors appraised studies independently, with disagreements resolved by discussion. Only one study (Byars et al. 2004) was assessed as high risk of bias and excluded from the analysis; this was because the name of the measurement tool was not provided and therefore could not be assessed for validity.

Outline of Included Studies

Eighteen studies met full inclusion criteria and were assessed to be of sound methodological quality via the appraisal.
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<table>
<thead>
<tr>
<th>Criteria 1. Selection</th>
<th>Two points</th>
<th>One point</th>
<th>No points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Truly representative of the average in the target population (all subjects or random sampling)</td>
<td>Somewhat representative of the average in the target population (non-random sampling), Sample size justified and satisfactory</td>
<td>Selected group of users</td>
<td>No description of the sampling strategy, Sample size not justified</td>
</tr>
<tr>
<td>Criteria 2. Measurement</td>
<td>Validated measurement tool</td>
<td>Non-validated measurement tool, but the tool is available or described</td>
<td>Interview/assessment conducted by a trained assessor (i.e., trained specifically for this study)</td>
</tr>
<tr>
<td>Criteria 3. Assessment</td>
<td>Interview conducted by a diagnostician (e.g., psychiatrist, psychologist) or diagnostician involved in the assessment process (e.g., overseeing assessment)</td>
<td>Interview/assessment conducted by a trained assessor (i.e., trained specifically for this study)</td>
<td>Information not clear</td>
</tr>
<tr>
<td>Criteria 4. Respondents</td>
<td>2 or more respondents (e.g., parent and teacher)</td>
<td>The statistical test used to analyse the data is clearly described and appropriate (e.g., confidence intervals, p value)</td>
<td>One respondent</td>
</tr>
<tr>
<td>Criteria 5. Statistical text</td>
<td>The statistical test is not appropriate</td>
<td>Not described or incomplete</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Assessment of bias of studies meeting full selection criteria

<table>
<thead>
<tr>
<th>Study</th>
<th>Criteria 1 Selection</th>
<th>Criteria 2 Measurement</th>
<th>Criteria 3 Assessment</th>
<th>Criteria 4 Respondents</th>
<th>Criteria 5 Statistical Text</th>
<th>Total score</th>
</tr>
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<tbody>
<tr>
<td>Andrews et al. (1999)</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>8</td>
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<tr>
<td>Angold et al. (2003)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Ashtma et al. (2001)</td>
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<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
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<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>7</td>
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<tr>
<td>Cailean et al. (1997)</td>
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<td>2</td>
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<td>0</td>
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<td>6</td>
</tr>
<tr>
<td>Cohen et al. (1995)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Costello et al. (2005)</td>
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<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>7</td>
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<tr>
<td>Engas et al. (2008)*</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
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<td>8</td>
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<tr>
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<td>6</td>
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<tr>
<td>Levent et al. (2008)</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>8</td>
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<tr>
<td>López-Villalobos et al. (2014)</td>
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<td>0</td>
<td>0</td>
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<td>5</td>
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<tr>
<td>Melina et al. (2014)</td>
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<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Menkovic et al. (2013)</td>
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<td>Nørrelyk et al. (2014)</td>
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<td>6</td>
</tr>
<tr>
<td>Park et al. (2014)</td>
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<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Simonoff et al. (1997)</td>
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<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>5</td>
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<td>Sugawara et al. (1999)</td>
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<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>6</td>
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<tr>
<td>Zwaan et al. (2007)</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>7</td>
</tr>
</tbody>
</table>

*Excluded from analysis due issues with methodological quality and measurement of ODD

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procedure, yielding 19 effect sizes for meta-analysis (Bird et al. 2006; contained two independent samples both with an independent effect size). Table 3 summarises the characteristics of each study. All studies were cross-sectional in design. The aggregated sample size was N = 44,107. Samples were from a broad range of countries, with the highest representation from the United States of America (USA; six studies). All samples had an average age range within middle childhood, however some included participants as young as five (Asher et al. 2001; Bird et al. 2006) and as old as 17-years-of-age (Angold et al. 2002). All measures used to determine the prevalence of ODD were based on DSM diagnostic criteria, and were responded to by parent and/or teacher and/or child via self-report measure and/or interview. The psychometric literature on these measures was examined by the first author in order to confirm that they were standardised, valid and reliable measures of child psychopathology.

Assessment of Publication Bias

Publication bias was assessed using a funnel plot in conjunction with Egger’s test of asymmetry (Egger et al. 1997). Funnel plots show the relationship between study level effect sizes and its standard error. The standard error

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Country/ Region</th>
<th>N</th>
<th>Age range of sample in years</th>
<th>Measures (Respondent)</th>
<th>Prevalence % Male/Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andria, Cala &amp; Gormez-Beneixo (1999)</td>
<td>Spain</td>
<td>387</td>
<td>10</td>
<td>KIDDS-SADS-E (Based on DSM-IV criteria) (Parent only)</td>
<td>4.9/2.3</td>
</tr>
<tr>
<td>Angold et al. (2002)</td>
<td>USA</td>
<td>920</td>
<td>9-17</td>
<td>The Child and Adolescent Psychiatric Assessment (CAPA, based on DSM-IV criteria) (Parent only)</td>
<td>2.4/1.2</td>
</tr>
<tr>
<td>Ashenfiet, Robel, Deute &amp; Allen (2003)</td>
<td>Ethiopia</td>
<td>1477</td>
<td>5-15</td>
<td>DICA (Based on DSM-III criteria) (Parent only)</td>
<td>12.1/6</td>
</tr>
<tr>
<td>Bird et al. (2008; USA)</td>
<td>USA</td>
<td>1158</td>
<td>5-13</td>
<td>DISC-IV (Based on DSM-IV criteria) (Parent &amp; Child)</td>
<td>6.6/2.9</td>
</tr>
<tr>
<td>Bird et al. (2006; PR)</td>
<td>Puerto Rico</td>
<td>1353</td>
<td>5-13</td>
<td>DISC-IV (Based on DSM-IV criteria) (Parent &amp; Child)</td>
<td>7.6/3.2</td>
</tr>
<tr>
<td>Carlson, Tamm &amp; Gaul (1997)</td>
<td>USA</td>
<td>2984</td>
<td>6-9</td>
<td>DSM-IV rating scale for ADHD and ODD (Teacher only)</td>
<td>4.2</td>
</tr>
<tr>
<td>Cohen et al. (1993)</td>
<td>USA</td>
<td>541</td>
<td>10-13</td>
<td>DISC-1 (Based on DSM-III-R criteria) (Parent &amp; Child)</td>
<td>14.2/10.4</td>
</tr>
<tr>
<td>Contello, Masrella, Fokkari, Kreier &amp; Angold (2003)</td>
<td>USA</td>
<td>6674</td>
<td>9-16</td>
<td>CAPA (Parent &amp; Child)</td>
<td>3.1/2.1</td>
</tr>
<tr>
<td>Knes et al. (2007)</td>
<td>The Netherlands</td>
<td>1317</td>
<td>6-8</td>
<td>ADHD (translated version of DICA; based on DSM-III criteria) (Parent only)</td>
<td>13.3/9.4</td>
</tr>
<tr>
<td>Leung et al. (2008)</td>
<td>China</td>
<td>541</td>
<td>Mean = 13.8; SD = 1.3</td>
<td>DISC-IV (Based on DSM-IV criteria) (Parent &amp; Child)</td>
<td>6.9/10.4</td>
</tr>
<tr>
<td>Lopez-Villalobos et al. (2014)</td>
<td>Spain</td>
<td>1069</td>
<td>6-16</td>
<td>Child Symptom Inventory (Based on DSM-IV criteria) (Parent only)</td>
<td>6.8/4.3</td>
</tr>
<tr>
<td>Mitta, Gerg &amp; Denis (2014)</td>
<td>India</td>
<td>900</td>
<td>6-11</td>
<td>Children’s Behaviour Questionnaire (Based on DSM-IV criteria) (Parent &amp; Child)</td>
<td>8.9/8.8</td>
</tr>
<tr>
<td>Munkvold, Lundvold &amp; Manger (2013)</td>
<td>Norway</td>
<td>707</td>
<td>7-9</td>
<td>Strengths and Difficulties Questionnaire (SDQ) (Parent &amp; Teacher)</td>
<td>4.1/3.4</td>
</tr>
<tr>
<td>Nielssen, Egele, Bauer, Klein &amp; von Gottsch (2014)</td>
<td>Germany</td>
<td>1676</td>
<td>Mean = 5.7</td>
<td>DISYTS-E (Based on DSM-IV criteria) (Parent only)</td>
<td>7.3/5.1</td>
</tr>
<tr>
<td>Park et al. (2014)</td>
<td>Korea</td>
<td>1465</td>
<td>6-12</td>
<td>DISC-IV (Based on DSM-IV criteria) (Parent only)</td>
<td>5.8/4.1</td>
</tr>
<tr>
<td>Simonoff et al. (1997)</td>
<td>USA</td>
<td>2762</td>
<td>8-16</td>
<td>CAPA (Based on DSM-III-R criteria) (Parent &amp; Child)</td>
<td>3.9/5</td>
</tr>
<tr>
<td>Sogawa et al. (1999)</td>
<td>Japan</td>
<td>114</td>
<td>7-9</td>
<td>Child Assessment Schedule (CAS; Based on DSM-III-R criteria) (Parent &amp; Child)</td>
<td>7.5/3.3</td>
</tr>
<tr>
<td>Zwin et al. (2007)</td>
<td>The Netherlands</td>
<td>2041</td>
<td>6-10</td>
<td>DISC-F (Based on DSM-IV criteria, Parent, SDQ (teacher SCICA (Based on DSM-IV criteria, Child)</td>
<td>13/9</td>
</tr>
</tbody>
</table>
is used to assess the precision of the effect size. Studies with larger standard errors provide a less precise estimate of the population parameter. Funnel plots indicate publication bias when the distribution of effect sizes is asymmetrical distributed around the weighted average effect size. This outcome indicates differences arise presumably because only significant (or even non-significant) findings were identified. A funnel plot showing the effect sizes included in the meta-analysis is presented in Fig. 2. Egger’s test did not reveal significant levels of asymmetry in the effect sizes (Intercept = -0.91, t(17) = 1.00, p = 0.33).

Meta-Analyses

A forest plot showing all 19 effect sizes and the weighted average is presented in Fig. 3. The average weighted RR was found to be 1.59 and statistically significant (RR = 1.59, 95% CI [1.36, 1.86], p < 0.001), indicating an averaged male:female prevalence ratio of 1.59:1. As recommended by Higgins et al. (2003), heterogeneity between effect sizes was assessed using the I² statistic. A moderate level of heterogeneity was found (I² = 55.67%). This suggests that 55.9% of differences between effect sizes represent true differences between those effect sizes and cannot be explained by chance.
As the male female prevalence ratio was lower than expected based on those found for other DBDs, it was decided to remove the two effect sizes that demonstrated trends in the opposite-to-expected direction (i.e., a higher prevalence in females than males; Ashenafi et al. 2001 & Leung et al. 2008) to explore how these outlier effect sizes may have attenuated the overall ratio. The subsequent meta-analysis found the male-female prevalence ratio rose to 1.75:1 and remained significant (RR = 1.75, 95% CI [1.52, 2.00], p < 0.001).

The fourteen effect sizes drawn from Western cultures (see Table 3) were analysed and found to have a statistically significant average weighted RR of 1.8 (RR = 1.8, 95% CI [1.55, 2.10], p < 0.001). There were no significant sex differences in prevalence in effect sizes drawn from non-Western cultures (RR = 1.08, 95% CI [0.76, 1.53], p > 0.05). The effect size for the sex difference in Western studies was found to be significantly larger than for non-Western studies (Q = 14.95, p < 0.002; see Fig. 4), demonstrating that the sex differences in ODD prevalence are more pronounced in Western than non-Western cultures.

Significant sex differences were found in the prevalence of ODD for studies published prior to 2000 (RR = 1.57, 95% CI [1.22, 2.02], p < 0.001) and studies published post the year 2000 (RR = 1.64, 95% CI [1.35, 2.00], p < 0.001). No significant difference was found between these two periods (Q = 14.36, p > 0.05; see Fig. 5).

**Discussion**

The primary purpose of the current meta-analysis was to provide a quantified male-female prevalence ratio for ODD during middle childhood in non-referred children. A systematic search of the literature yielded 19 independent effect sizes. The weighted average Relative Risk demonstrated significantly more boys are affected by ODD than girls, with a male:female prevalence ratio of 1.59:1 (which rose to 1.75:1 with the exclusion of two outlier studies). Two secondary aims were to explore the impact of Culture and Time on the male:female prevalence ratio. Significant sex differences were found in ODD prevalence in Western but not non-Western cultures, as well as in studies published prior to and post the year 2000; with sex differences in prevalence remaining consistent between these two time periods.

Overall, our results confirm ODD follows a similar pattern to other DBDs in its prevalence, in that boys are more commonly affected than girls, but only in Western cultures. However, the sex difference found in the current results does not appear to be as pronounced as those reported in meta-analytic reviews for ADHD and CD (cf. ratios of 2.2:1 to 3:1:1 and 2.4:1 to 3:1 for ADHD and CD respectively; Enkovaara et al. 2013; Willcutt 2012; Witteken et al. 2011). Further, our overall male:female prevalence ratio of 1.59:1 is not reflective of the ratio previously reported for the child-onset developmental pathway of antisocial behaviour (10:1; Moffitt and Caspi 2001), and instead appears almost identical to the adolescent-onset pathway (1.5:1; Moffitt and Caspi 2001). These discrepancies warrant discussion. One possible explanation relates to the diagnostic characteristics of DBDs. ODD diagnostic criteria encompass mood problems (e.g., loses temper, touchy or easily annoyed, angry or resentful), argumentativeness and defiance (e.g., argues with authority figures, defies or refuses to comply with requests or rules) and vindictiveness (APA 2013). These domains may in fact reflect attentional- and/or mood-oriented symptoms, which are inherently different from the action- and/or activity-oriented symptoms of CD and ADHD (excluding the ADHD inattentive subtype [ADHD-I]; e.g., physical fighting, physical cruelty, sexual behaviour, destruction of property for CD, and problems with activity such as fidgeting, difficulty remaining seated, and inappropriate running and climbing for ADHD). Previous research has shown boys are more likely to
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display these externally-directed behaviours than girls, while
tend to exhibit internally-directed symptomology, such as
mood lability, shyness, withdrawal and hypersensitivity more
so than boys (Carlson et al. 1997; Kann and Hanna 2000;
Lahey et al. 2000; Tiet et al. 2001). Given the predominant
presence of internalising-type symptomology in ODD, it
might not be surprising that girls may be more prone to
experiencing ODD than other DBDs, and/or boys less so.
Examination of sex differences in ADHD-I adds support to
this hypothesis. ADHD-I includes features such as inattention,
distractibility and difficulties with concentration and memory
(APA 2013), which, similar to ODD, are less action- and
activity-orientated than other ADHD subtypes and CD.
Paired with this, the male-female prevalence ratio of ADHD-
I is reported as lower than for ADHD-C (2.2:1 cf 3.6:1 respec-
tively, Willcutt 2012), giving weight to the notion that action-
and activity-orientated symptoms in DBDs may be less com-
mon in girls than boys.

Our finding that sex differences in ODD prevalence were
only significant in Western countries is particularly interesting
given the overall prevalence of ODD is found not to vary
across cultures (Canino et al. 2010). Thus, the lack of sex
differences in non-Western cultures, when compared to the
significant sex differences found in Western cultures, may be
due to either (i) a decreased prevalence of ODD in boys
from non-Western cultures, or (ii) an increased prevalence of ODD
in girls from non-Western cultures. If the latter of these two
is the case, this supports the hypothesis presented earlier that
cultural differences in sex roles may be influential. Non-
Western cultures where sex roles are more traditional may
have a lower threshold for ODD behaviours in girls. Thus,
subclinical ODD behaviours demonstrated by girls in
Western cultures might be viewed as pathological in Non-
Western cultures if they violate culturally derived sex-role
stereotypes. This may lead to higher rates of referral and di-
agnosis of girls in non-Western cultures compared to Western
cultures. Conversely, Western cultures may have increased
tolerance for disruptive behaviours in girls (perhaps due to a
higher tolerance of violations of gender norms) resulting in a
lower risk of referral and diagnosis. Both of these interpreta-
tions would impact on prevalence rates in girls. Further inves-
tigations into why sex differences occur in Western but not
non-Western cultures, despite overall: prevalence rates not
varying between the two (Canino et al. 2010), are needed.

Our results that a significant sex difference occurs in ODD
prevalence, albeit only in Western cultures, invites speculation
about why this difference exists and about the risk and/or
protective factors associated with ODD. For example, cultural
differences in prevalence-by-gender rates might suggest po-
tentially modifiable socio-cultural factors that influence the
epidemiology, referral, and/or diagnosis of ODD in boys and/or
girls. Further, the notion of sex-specific ODD diagnostic
criteria to ensure appropriate diagnosis of both genders is
worthy of exploration, given the hypothesis that ODD may
manifest differently in boys and girls (Crick and Zahn-Waxler
2003; Wachbusch and King 2006). The possibility of an un-
der diagnosis of girls, due to presentations that might not
match current diagnostic criteria as demonstrated by
Wachbusch and King (2006), may help to account for girls’
lower ODD prevalence. Factors hypothesised to increase
boys’ risk of DBDs also need further study. In particular boys’
greater exposure to less-than-ideal parental practices is an
important avenue as (i) parenting practices are particularly
predictive of oppositional and aggressive behaviours in chil-
dren (Stomrakshak et al. 2000) and (ii) parenting practices rep-
resent a potentially modifiable influence.

We failed to find evidence of changes in sex differences in
ODD prevalence in investigations published prior to and post
the year 2000. Across both time periods prevalence rates were
higher in boys. It is possible this was due to the year chosen
to split groups, however inspection of Fig. 3, in which effect
sizes are plotted chronologically, does not suggest an alterna-
tive split point, or evidence of time-related patterns. This find-
ing does not fit with Loebel et al. (2000) who suggested in-
creases in girls’ delinquency over time might have attenuated
sex differences in externalising behaviours. Instead, this in-
crease may have a greater impact on sex differences in CD
prevalence given delinquent behaviours are more symptomat-
ic of CD than ODD. The finding that sex-differences in ODD
are consistent across time in the face of cultural and societal
changes lends weight to biological accounts for the aetiology
of ODD. Thus, future research into the biological aetiology of
ODD and other externalising disorders should ensure they
consider sex as a variable of interest in their investigations.
Environmental factors (such as attitudes on gender) cannot be
ruled out as a potential influence, as these have also remained
somewhat stable in recent decades (Bolzendahl and Myers
2004; Sayer 2005; Twenge 1997).

Limitations

The authors acknowledge several limitations of the cur-
rent study. Despite our comprehensive search strategy it is
possible that literature was missed for various reasons
(e.g., uncommon search/key terms). There was also little
consistency between included studies in their measures
used and the number of respondents. This may have led
to differences in the ODD construct being measured,
impacting the validity of our results. Various studies en-
gaged individual, dyad or triad respondents. Although it
might be assumed that more respondents would increase
the accuracy of estimates, there is often little consistency
in ratings between multiple respondents, affecting the ac-
curacy of combined ratings (O’Neill et al. 2014; Stokes
et al. 2014). This issue may help to account for the moder-
ate level of heterogeneity of our results.
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Only eight of the 19 effect sizes included here reported significant sex differences in the prevalence of ODD. Given this inconsistency we attempted to identify methodological parameters that might distinguish the studies that found significant differences from those that did not find significant differences. Data summarised in Table 3 were examined for trends, including geographical location of the sample (including cultural considerations), age range of the participants, sample size, measures used, and number of respondents. No obvious trends or patterns were discernible.

Conclusion

The current review found an overall male dominance in ODD prevalence, with this result holding across Western, but not non-Western, cultures and across time. Attention should now be paid to investigating the possible reasons for these sex differences in ODD prevalence from a bio-psycho-social perspective. Several hypotheses have been suggested here and warrant future investigation, particularly the notion of cultural differences in prevalence-by-gender rates, gender-biased diagnostic criteria, and the role of gender differences in early ODD risk factors. Given that ODD is a predictor of later psychopathology, such as CD and personality disorders (Burke et al. 2002; Holmes et al. 2001; Loebel et al. 1995; Maughan et al. 2004), ensuring that the male dominance is not due to under- or misdiagnosis of ODD in females’ early in development is critical to minimize their risk of lifelong detrimental effects.

Compliance with Ethical Standards

Funding There was no funding for this study.

Conflicts of Interest David H. Demerar declares he has no conflict of interest. Marilyn Hoeky declares she has no conflict of interest. Jane A. McGillis declares she has no conflict of interest. Jarrad A. G. Lunn declares he has no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

This article does not contain any studies with human participants performed by any of the authors. For this type of study formal consent is not required.

References


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Appendix B

Study 3 published version

The Influence of Child Gender on the Prospective Relationships between Parenting and Child ADHD

David H. Demmer 1,7,8 & Francis Paolino 3,4; Mark A. Stoles 5,6; & Jane A. McGillivray 5,6 & Merrilyn Hooley 5,6

Abstract. The aims of the current study were to (i) explore the potential bidirectional, prospective relationships between parenting and child ADHD, and (ii) explore whether these relationships differed on the basis of child gender. Data were obtained from waves 1 (children aged 4 to 5 years) to 5 (children aged 12 to 13 years) of the Longitudinal Study of Australian Children (LSAC) dataset (child cohort). In order to examine dimensions of both mothers' and fathers' parenting, a subsample of nuclear families with mothers, fathers and children present at all waves was extracted (final sample = 1932, sons = 881, daughters = 851). Child ADHD measures included the hyperactive-impulsive subscale of the Strengths and Difficulties Questionnaire for symptoms, and parent-report questionnaire for diagnoses. Mothers and fathers completed scales on dimensions of Angry, Warm and Consistent Parenting. A cross-lagged panel model demonstrated (i) higher child ADHD symptoms at wave 1 led to a global increase in less-than-optimal parenting at wave 2, and (ii) child ADHD symptoms and Angry Parenting share a prospective, bi-directional relationship (whereby increases in one predicted increases in the other over time) during earlier years of development. Latent growth curve models demonstrated that increases in Angry Parenting across time were significantly predicted by increases in child ADHD symptoms. A logistic regression demonstrated that both mothers' and fathers' Angry Parenting at wave 1 significantly predicted an ADHD diagnosis in children at wave 3. No predictive relationships differed between child genders; thus, it appears these prospective pathways are similar for both sons and daughters.

Keywords: Childhood psychopathology - ADHD - Externalizing disorder - Parent Gender

Electronic supplementary material: The online version of this article (doi:10.1007/s10803-017-0264-7) contains supplementary material, which is available to authorized users.

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Published online: 02 March 2017
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diagnosis of ADHD tend to have parents who are less warm and less involved (Ellis and Nigg 2009; Tripp et al. 2007), less consistent in their punishment (Causen et al. 2012), and/or more overprotective (Chang et al. 2013) and controlling (Rogers et al. 2009) than the parents of their typically-developing peers. Higher levels of child ADHD symptoms are also related to higher levels of maternal hostility (Harold et al. 2013), parental stress (Graziano et al. 2011), as well as various other aspects of negative parenting (e.g., poor monitoring/supervision, inconsistent discipline and corporal punishment; Haack et al. 2016).

Despite the wealth of investigations into the relationships between various aspects of parenting and child ADHD, several important questions remain unanswered. First, as most previous investigations have been cross-sectional in design, the causal direction of the association between parenting and child ADHD (i.e., whether certain parenting dimensions are predictive of, or an outcome of, child ADHD, or if a bi-directional relationship exists) remains unclear. Second, despite suggestions that child gender may moderate the relationship between parenting and child ADHD (Braza et al. 2015; Johnston and Mash 2001), this notion is yet to be formally tested (Lifford et al. 2008).

Two longitudinal studies have explored the prospective relationships between parenting and child ADHD (Keown 2012; Lifford et al. 2008) in attempts to address the question of causality. The first, Lifford et al., examined the notion of bi-directionality and found different results for mothers and fathers. For mothers, higher levels of child ADHD symptomatology predicted higher levels of maternal rejection over a 12-month period, while the reverse relationship was found for fathers, with higher levels of paternal rejection predicting increases in child ADHD symptoms in children over the same period. The second, Keown (2012), examined unidirectional relationships only, and again found different relationships for mothers and fathers. Results demonstrated that higher levels of paternal sensitivity and positive regard, and higher levels of maternal positive regard, predicted lower levels of ADHD symptoms in sons across a 2 1/2 year period. Further, lower levels of maternal warmth predicted higher levels of sons’ ADHD symptoms over the same period.

The findings of Lifford et al. (2008) and Keown (2012) contribute to the understanding of the prospective relationship between parenting and child ADHD, however several limitations exist with these investigations that necessitate further work in this area. First, both studies called for investigations utilising larger sample sizes to strengthen the confidence of findings. Second, Keown was limited by investigating only unidirectional relationships (earlier parenting predicting later child ADHD), which precluded the potential identification of alternate, or bi-directional, relationships. This is a notable omission given common developmental models of ADHD (e.g., Johnston and Mash 2001), and the findings of Lifford et al., suggest bi-directional relationships likely exist between parenting and child ADHD. Third, both studies omit child gender as a potential moderator despite the potential importance in examining its effect on the relationship between parenting and child ADHD (Braza et al. 2015; Johnston & Mash).

Gender, specifically male gender, is a known risk factor in the development of ADHD, as demonstrated by the higher prevalence of ADHD in boys when compared to girls (approximately three boys to every one girl in community samples; Erskine et al. 2013; Wilcutt 2012). However, as ADHD research typically involves male-only samples, the way that child gender operates as a risk factor in the development of the disorder remains unclear (Johnston and Mash 2001). This may have important implications in the treatment of ADHD, particularly if different pathways of disease occur for boys and girls. For example, parent training is an important component of most psychosocial treatments for child ADHD (National Health and Medical Research Council 2012) based on the notion that parenting impacts child ADHD. Therefore, if parenting is differentially related to ADHD for girls and boys then current interventions may not be equally effective for both genders if the current ‘one-size-fits-all’ approach to treatment is taken.

Although the notion of whether the prospective relationships between parenting and ADHD differ between daughters and sons is yet to be examined, parallel areas of research suggest a gender difference may exist. For example, it has consistently been demonstrated that parents commonly engage in different parenting behaviours with sons compared to daughters. Sons tend to receive higher levels of authoritarian parenting (e.g., corporal punishment, lack of explanation about punishment, verbal hostility) and less positive parenting (e.g., less displays of warmth, less aware and responsive to cues from the child) than girls (Barnett and Scarzella 2013; Russell et al. 1998). Sons also receive fewer displays of emotional understanding (Jivash et al. 2000), praise, and physical affection than daughters, yet receive higher rates of yelling and smacking (Lloyd and Devine 2006). Given that (i) many of these less-than-optimal parenting behaviours have been linked to ADHD, and (ii) child gender is related to both parenting and ADHD, it might be that child gender also impacts the relationship between parenting and ADHD. For example, theory (e.g., Keenan and Shaw 1997) as well as previous research (e.g., Wright et al. 2013) suggests that the socialisation of gender often involves a greater tolerance of internalising behaviours in girls and externalising behaviours in boys by socialising agents (e.g., parents). These socialising influences potentially attenuate the relationship between negative parenting and externalising behaviours in girls, and in boys potentially contribute to the development of a maladaptive cycle of less-than-optimal parenting influencing externalising behaviours, which go on to influence less-than-optimal parenting. It might therefore be expected that less-
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than-optimal parenting might have a greater influence on the
development of externalizing behaviours (such as those
demonstrated in ADHD) in boys than in girls.

The Current Study

The aims of the current study were twofold: first, to
explore the potential bidirectional, prospective rela-
tionships between parenting dimensions and child ADHD,
and second, to explore whether these relationships dif-
fered on the basis of child gender, in a large sample of
Australian nuclear families. Nuclear families were inves-
tigated due to previous research often focusing only on
mothers’ parenting. This focus, however, overlooks the
important influence of fathers’ parenting on child behav-
ior. As evidence suggests that child developmental out-
comes differ depending on whether maternal or paternal
influences are considered (Brazel et al. 2015; Lamb
2004), and based on previous longitudinal research in
this area demonstrating that mothers’ and fathers’ parent-
aging are differentially associated with child ADHD
(Kown 2012; Lifford et al. 2008), it is important to
consider both parents in investigations. The current study
tested a number of hypotheses. Bi-directional relation-
ships were expected based on arguments by common
developmental models of ADHD (e.g., Johnston and
Mash 2001). Gender differences in these relationships
were also expected based on previous theory (Keenan
and Shaw 1997) that suggests girls might be discouraged
from presenting their difficulties in an externalizing man-
ner. It was hypothesized that:

1. Bi-directional, prospective relationships will exist be-
tween parenting dimensions and child ADHD symptoms.
Specifically, higher levels of less-than-optimal parenting
(i.e., higher scores on Anxious Parenting, lower scores on
Consistent Parenting, and lower scores on Warm
Parenting) will be predictive of, and predicted by, higher
levels of ADHD symptoms in boys and girls across time
2. The predictive relationship between parenting dimensions
and child ADHD symptoms will be stronger for boys than
for girls, as measured by chi square change between gender-
specific models
3. Less-than-optimal parenting (i.e., higher scores on Anx-
ious Parenting, lower scores on Consistent Parenting, and
lower scores on Warm Parenting) at wave 1 will be predictive
of an ADHD diagnosis in boys and girls at wave 3
4. The predictive relationship between parenting dimensions
and ADHD diagnosis will be stronger for boys than girls
as measured by chi square change between gender-
specific models

Method

Participants

Data for this study were obtained from waves 1 through 5 of
the Longitudinal Study of Australian Children (LSAC). A
comprehensive overview of the LSAC sampling design, data
collection methods and measures have been described else-
where (Australian Institute of Family Studies 2013; Soltwe-
et al. 2005; Zahn et al. 2014), thus only a brief outline is
provided here. A two-stage cluster sampling design was used
to recruit two cohorts, an infant cohort (children 3–19 months
of age at wave 1) and a child cohort (children 4–5 years of age
at wave 1). First, stratification occurred at the state of resi-
dence level, and urban versus rural level. Postcodes (exclud-
ing the most remote) were then sampled. Second, all children
from sampled postcodes who were born between March 2003
and February 2004 (infant cohort), and March 1999 and
February 2000 (child cohort), and enrolled in the Australian
Medicare Database, which is the most comprehensive data-
base of Australia’s population, were contacted. Final samples
were 5107 for the infant cohort and 4983 for the child cohort.
Only the child cohort was used in the current study due to
child ADHD measures being present from wave 1 for this
cohort but not for the infant cohort. In order to explore
dimensions of both maternal and paternal par-
tering, a subsample of the original 4983 child cohort
cases was extracted. Cases were included in the current
sample if mother, father and child were present, and
participated, through all waves, with a final subsample of
1932, 951 daughters (49.2%) and 981 sons (50.8%).

As causal pathways between non-referral and referred
cases are considered fundamentally different, all referred cases
(i.e., children with a diagnosis of ADHD) were removed from
the cross-lagged path analysis and the latent growth curve
models (LGCMS) as per appropriate epidemiological method-
ology (Rothman et al. 2008). However, parallel cross-lagged
and LGCMS analyses were conducted with the full sample (i.e.,
referred and non-referred cases) with no difference in path-
ways found between the models. Referred cases were includ-
ed in the logistic regression as child ADHD diagnosis was the
dependent variable in this analysis.

Measures

ADHD

Child ADHD symptoms were measured in the LSAC dataset
via the strengths and difficulties questionnaire (SDQ; Cronbach’s α = 0.88). Mothers’ SDQ scores were used in
the current study as mothers had lower levels of missing data
on this measure compared to fathers. The SDQ is a brief be-
havioural screening questionnaire for children 4- to 16-years

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of age, consisting of 25 items covering behavioural and emotional problems, and prosocial behaviours (Goodman 1997), and contains five subscales. The hyperactivity-inattention subscale is made up of five items (e.g., “Restless, overactive, cannot stay still for long” and “easily distracted, concentration wanders”). Items on the subscale are rated on a three-point index: 0 (not true), 1 (somewhat true), or 2 (certainly true), with a possible range of scores from 0 to 10. Higher scores indicate higher levels of hyperactive-inattentive symptoms. The validity and sensitivity of the SDQ as a screening tool for ADHID symptoms has been well established (Goodman et al. 2000; Stone et al. 2010), including in Australian samples (Hawes and Dadds 2004). For example, Hawes and Dadds used factor analytic techniques to investigate the validity and reliability of the SDQ (parent-report) on an Australian community sample of 1359 boys and girls 4- to 9-years of age. Moderate to strong internal reliability and stability was found across all subscales, suggesting the SDQ is a valid and reliable measure of both behavioural and emotional symptomology in Australian children.

Child ADHD diagnosis in the LSAC dataset was determined by a single item parent-report question with a yes or no response (“Does child have any of these ongoing problems? ADD or ADHD?”). A second question regarding ADHD medication use (“Has your child ever taken any medication for attention deficit disorder or ADHD?”) was used in the current study to validate the response (83% of parents who reported “yes” to ADD or ADHD as an ongoing problem also reported their children had been prescribed ADD or ADHD medication at wave 5).

Parenting Dimensions

The parenting dimensions extracted from the LSAC dataset were Angry Parenting, Warm Parenting and Consistent Parenting. These dimensions have previously demonstrated associations with child ADHD (angry; Keown and Woodward 2002; warm; Chang et al. 2013; Keown 2012; consistent: Ellis and Nigg 2009). The construct validity and measurement quality of these scales, as they relate to the LSAC dataset, have been published in a technical paper (Zurbrick et al. 2014). For the current study, very good internal consistencies were found for all scales (mothers’ Angry Parenting α = 0.86, fathers’ Angry Parenting α = 0.85; mothers’ Consistent Parenting α = 0.88, fathers’ Consistent Parenting α = 0.86; mothers’ Warm Parenting α = 0.88; fathers’ Warm Parenting α = 0.85). Each scale contained five items with responses provided on a 5-point scale (1 = never/ almost never, 2 = rarely, 3 = sometimes, 4 = often, 5 = always/ almost always). Both Angry Parenting and Consistent Parenting were measured using items from scales in the National Longitudinal Survey of Children and Youth (NLSYC; Statistics Canada 2000), while Warm Parenting was measured using a modified scale from the Child Depression Questionnaire (Patterson and Sanson 1999).

Angry Parenting measured parents’ use of aversive or harsh discipline via items regarding feelings of anger or frustration towards the child, as well as emotional reactivity (e.g., “How often are you angry when you punish this child?”), with higher scores on this measure representing less praise, more disapproval, and more negative emotions directed towards the child. Parents of children with ADHD have been demonstrated to be more angry and mean in their disciplinary encounters with their children than parents of non-ADHD children (Keown and Woodward 2002). Further, harsh discipline and/or hostility directed towards children has been shown to influence the development and maintenance of behavioural problems during childhood (Chang et al. 2003); while decreases in these parenting behaviours leads to positive change in child behaviour (Sanders et al. 2000).

Warm Parenting measured the amount of warmth and affection displayed towards the child (e.g., “How often do you hug or hold this child for no particular reason?”), with higher scores on this measure representing more displays of warmth and affection. Children with ADHD regularly receive less warmth (Chang et al. 2013), with lower levels of parental warmth predicting poorer developmental outcomes for children (Davidov and Grusec 2006) and substance use disorders in children with ADHD once they reach adolescence (Tandon et al. 2014).

Consistent Parenting measured the setting and consistent application of age-appropriate rules and expectations (e.g., “How often does this child get away with things that you feel should have been punished?”). Higher scores on this measure represented the more consistent setting and application of rules and expectations. Inconsistent parenting has been shown to strongly contribute to children’s problem behaviours, and is one of the key areas addressed in behavioural family interventions (Sanders et al. 2000).

Statistical Methods

In order to conduct a comprehensive investigation, a series of analyses were selected and conducted to test the hypotheses of the current study and to address different aspects of prospective relationships between parenting and (i) child ADHD symptoms, and (ii) child ADHD diagnosis. A cross-lagged panel model, two latent growth curve models (LGCMs), as well as a logistic regression, were used to test the hypotheses. All analyses were performed using Mplus version 7.2 and SPSS version 23. Standard cut-offs for fit indices were used: CFI and TLI > 0.95 for excellent fit and ≥0.90 for acceptable fit and RMSEA ≤ 0.05 for good fit (Brown and Cudeck 1993; MacCallum et al. 1996; Steiger 1989). Socio-economic status (SES) was controlled for within all analyses as SES has been shown to be an important associate for both parenting...
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(Friedson 2016; Pinderhughes et al. 2000) and child ADHD (Russell et al. 2015). The SES variable was a single continuous score calculated based on (i) combined family income, (ii) educational attainment of both parents, and (iii) parents’ occupational status. This score therefore represented the social and economic resources available to the family. Rates of missing data for all variables ranged from approximately 0.1% to 8.3% for individual variables across waves. Dimensions of fathers’ parenting generally had the highest rates of missing data across all waves. Missing data were imputed using multiple imputation in MPLUS.

Cross-Lag Panel Model

A cross-lagged panel analysis using continuous manifest variables was conducted to test Hypothesis 1. Bi-directional effects examine the reciprocal relationship between two constructs measured across time, and addresses whether a particular construct, measured at a particular time point, is predictive of change in another variable at a later time point. In our model, variance in each of the variables of interest (parenting dimensions and child ADHD symptoms) was predicted from two main sources: autoregressive paths and cross-lagged paths. The autoregressive paths represent the effect of the construct on itself across time (e.g., child ADHD symptoms at time 1 predicting child ADHD symptoms at time 2; Selig and Little 2012). The inclusion of autoregressive paths minimizes bias in the estimation of cross-lagged paths (Cole and Maxwell 2003; Gullone and Reichardt 1987; Selig & Little). The cross-lagged paths represent the relationships of interest (e.g., the variance in child ADHD at time 2 that is predicted by parenting dimensions at time 2). Given the inclusion of the autoregressive paths, the only variance available for prediction by the cross-lagged paths is the residual variance in the outcome variable, thus providing a more sensitive analysis.

Latent Growth Curve Models

Latent growth curve models were also used to test Hypothesis 1. LGCMs assess whether initial levels of one variable (known as the intercept), or the change trajectory (known as the slope) in that variable over time, predicts the change trajectory (slope) of another variable over time. The value of the LGCM is that it establishes how different responses emerge across time, accounting for how earlier decisions influence later outcomes. Further, this enables an understanding of how subtle differences at a given time point may lead to considerable differences in outcome (i.e., sensitive dependence upon initial conditions). Two separate LGCMs were constructed to assess the relationships between the intercept and slope of a predictor variable on the slope of an outcome variable. In the first LGCM, the intercepts and slopes of mothers’ and fathers’ Consistent, Angry and Warm Parenting were predictors for the slope of child ADHD symptoms. In the second LGCM, the intercept and slope of child ADHD symptoms were the predictor variables, with the slopes of mothers’ and fathers’ Consistent, Angry and Warm Parenting as outcomes.

Logistic Regression

A logistic regression analysis was used to test Hypothesis 3, and examined if mothers’ and fathers’ Consistent, Angry and/or Warm Parenting at Wave 1 predicted a child ADHD diagnosis at wave three. Wave 1 was selected as the prediction wave as the cross-lagged model demonstrated that this was the time point where parenting and child ADHD symptoms were most related. Wave 3 was selected because children at this age (6–9 years of age) are at the average age of child ADHD diagnosis (Keshler et al. 2005).

Structural Invariance Testing

Structural invariance testing was conducted for the cross-lag panel model and LGCMs to test Hypothesis 2, and also for the logistic regression to test Hypothesis 4 (except where statistical significance was not found in the relationships of interest), to determine whether the predictive relationships in these models were equivalent across child genders. First, an unconstrained model was tested for boys and girls separately, in which parameters were free to vary. Second, the model was constrained whereby the regression paths for each gender were constrained to be equal. The paths for both models were then compared. Structural invariance (i.e., the models were different based on child gender) was determined if Delta chi-square ($\Delta \chi^2$) significantly differed from zero ($p < 0.05$).

Results

Preliminary Analyses

Table 1 presents the means and standard deviations (SDs) of parenting dimensions and child ADHD symptoms for non-referred and referred children separately, and summarises the results of t-tests (significant differences and Cohen’s D effect sizes) comparing non-referred and referred children on parenting dimensions and child ADHD symptoms. Table 2 presents the means and SDs of parenting dimensions and child ADHD symptoms for non-referred girls and boys separately, and summarises the results of t-tests (significant differences and Cohen’s D effect sizes) comparing girls and boys on parenting dimensions and child ADHD symptoms. Mothers’ and fathers’ Angry Parenting was higher in (i) referred children compared to non-referred children and (ii) non-referred boys compared to non-referred girls across all five waves. From
wave 3, referred children received significantly less Warm Parenting from their mothers than non-referred children. This same difference was found for fathers' Warm Parenting in waves 1 and 3. Mothers' Warm Parenting was higher for daughters than for sons in waves 4 and 5, and fathers' Warm Parenting were higher for daughters than sons from wave 2 onwards. Referred children had significantly higher ADHD symptoms than non-referred children across all waves (see Table 1). Further, non-referred boys had significantly higher child ADHD symptoms than non-referred girls across all waves (see Table 2).

Table 3 presents the rates of ADHD diagnosis across waves for girls and boys, as well as significant differences and effect sizes. Across all waves, boys were significantly more likely than girls to have an ADHD diagnosis.

Cross-Lagged Panel Model

First, a baseline control model was run containing only the autoregressive pathways: $\chi^2(571, N = 1932) = 2464.38$. The pathways of interest were then added to the model (i.e., the prospective, bidirectional pathways between parenting dimensions and child ADHD symptoms): $\chi^2(523, N = 1932) = 2157.99$. The addition of the pathways of interest significantly improved the model $\chi^2(48, N = 1932) = 306.39, p < 0.001$, demonstrating that the pathways of interest were important additions to the model and predicted significant variance in the model over and above the control autoregressive model.

Good fit for the cross-lagged model examining the bi-directional effects of parenting dimensions and child ADHD was found (RMSEA = 0.04, CFI = 0.96, TFI = 0.94). For simplicity, Fig. 1 presents just the significant findings regarding the relationships of interest (i.e., the bi-directional prospective relationships between parenting dimensions and child ADHD symptoms) as well as the significant autoregressive pathways of the connecting time points. The full table of results is available by request from the corresponding author. Overall, mothers’ and fathers’ Angry Parenting and child ADHD symptoms demonstrated a prospective, bi-directional relationship; however this was only significant in the early years (ages 4 to 7 years). Further, this
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Table 2  Means and standard deviations for, and significant differences and effect sizes (Cohen’s D) between, non-referred girls and boys across waves

<table>
<thead>
<tr>
<th></th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
<th>Wave 4</th>
<th>Wave 5</th>
<th>Wave 1</th>
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<th>Wave 3</th>
<th>Wave 4</th>
<th>Wave 5</th>
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<td>2.08</td>
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<td>2.00</td>
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<td>(0.57)**</td>
<td>(0.22)</td>
<td>(0.24)</td>
<td>(0.24)</td>
<td>(0.22)</td>
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<td>2.02</td>
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<td>(0.59)</td>
<td>(0.62)</td>
<td>(0.57)**</td>
<td>(0.27)</td>
<td>(0.27)</td>
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<td>4.32</td>
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<td>(0.57)**</td>
<td>(0.13)</td>
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<td>4.06</td>
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<td>(0.67)</td>
<td>(0.59)**</td>
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<td>0.55</td>
<td>0.55</td>
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</tr>
<tr>
<td>(0.51)</td>
<td>(0.59)</td>
<td>(0.59)</td>
<td>(0.61)</td>
<td>(0.60)</td>
<td>(0.60)</td>
<td>(0.59)**</td>
<td>(0.15)</td>
<td>(0.15)</td>
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<tr>
<td>Father consistent parenting</td>
<td>4.04</td>
<td>4.14</td>
<td>4.14</td>
<td>4.13</td>
<td>4.08</td>
<td>4.04</td>
<td>0.66**</td>
<td>0.63</td>
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<td>(0.64)</td>
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<td>(0.64)</td>
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<tr>
<td>ADHD symptoms</td>
<td>2.61</td>
<td>2.62</td>
<td>2.53</td>
<td>2.46</td>
<td>2.42</td>
<td>2.48</td>
<td>0.29**</td>
<td>0.84</td>
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<td>(1.98)</td>
<td>(1.90)</td>
<td>(1.84)</td>
<td>(1.93)</td>
<td>(1.80)</td>
<td>(1.80)</td>
<td>(1.93)**</td>
<td>(0.55)</td>
<td>(0.55)</td>
<td>(0.55)</td>
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</table>

t-tests compared girls to boys for wave and parenting dimension. E.g., Wave 1 mother angry parenting for girls compared to Wave 1 mother angry parenting for boys. Significances and effect sizes relate to these comparisons.

α = t-test non-significant therefore no effect size calculated
* p < 0.05
** p < 0.01
*** p < 0.001

Relationship appeared more continuous over time for fathers than mothers. In terms of uni-directional effects, higher levels of child ADHD symptoms predicted higher levels of mothers’ and fathers’ Angry Parenting throughout all waves. In addition, higher levels of child ADHD symptoms predicted decreases in Warm Parenting at wave 2 for fathers and at wave 3 for both mothers and fathers. Higher child ADHD symptoms at preceding waves also predicted less Consistent Parenting at wave four for mothers and wave five for fathers. Increases in mothers’ Consistent Parenting at wave three also predicted decreases in child ADHD symptoms at wave four.

Structural invariance testing indicated the cross-lag model did not differ based on the gender of the child (Δχ² p > 0.05).

Latent Growth Curve Models

Two separate LGMs were conducted. The paths specified in the LGMs were identical to the cross-lagged path model however also included the intercept and slope for ADHD symptoms and each parenting dimension for both mothers and fathers. Model one, where the intercept and slope of mothers’ and fathers’ Consistent, Angry and Warm Parenting were predictors, demonstrated good fit (RMSEA = 0.02, CFI = 0.99, TLI = 0.99). Model one results demonstrated that no parenting dimension significantly predicted change in child ADHD symptoms over time. Due to this null finding, no invariance testing for child gender was assessed.

Table 3  Raw of ADHD diagnosis, significant differences and effect size (phi), for girls and boys across waves

<table>
<thead>
<tr>
<th></th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
<th>Wave 4</th>
<th>Wave 5</th>
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<tr>
<td>Girls</td>
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<td>0.009%</td>
<td>0.009%</td>
<td>0.009%</td>
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<tr>
<td>%</td>
<td>0.09%</td>
<td>0.09%</td>
<td>0.09%</td>
<td>0.09%</td>
<td>0.09%</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Boys</td>
<td>10**</td>
<td>17**</td>
<td>34***</td>
<td>55***</td>
<td>45***</td>
</tr>
<tr>
<td>%</td>
<td>0.02%</td>
<td>0.07%</td>
<td>0.46%</td>
<td>0.57%</td>
<td>0.59%</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Phi</td>
<td>0.07</td>
<td>0.07</td>
<td>0.11</td>
<td>0.11</td>
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</tbody>
</table>
| Ratio   | boy/girl ratio

** p < 0.01
*** p < 0.001

Ratio = boy/girl ratio
conducted. Model two, where the intercept and slope of child ADHD symptoms were the predictors of the slope of mothers' and fathers' Consistent, Angry and Warm Parenting were outcomes, also demonstrated good fit (RMSEA = 0.02, CFI = 0.99, TLI = 0.99). Model two results indicated that increases in child ADHD symptoms over time significantly predicted increases in both mothers' and fathers' Angry Parenting, as well as decreases in mothers' Warm Parenting, over time. Child ADHD did not significantly predict any other parenting dimensions. Structural invariance testing demonstrated these relationships were not significantly different based on the gender of the child ($\Delta \chi^2 = p > 0.05$).

**Logistic Regression**

The results of the logistic regression demonstrated that mothers' and fathers' Angry Parenting at wave one significantly predicted a child ADHD diagnosis at wave three. No other parenting styles were significant predictors of a child ADHD diagnosis. Structural invariance testing demonstrated these relationships were not significantly different based on the gender of the child ($\Delta \chi^2 = p > 0.05$). The full table of results can be found in the online supplementary material.

**Discussion**

The aim of the current study was to explore the potentially bidirectional prospective relationships between parenting and child ADHD, and to examine if these relationships varied as a function of child gender. Partial support was found for Hypothesis 1, that predicted bi-directional prospective relationships between less-than-optimal parenting and child ADHD symptoms, with significant prospective, bi-directional relationships found between mothers' and fathers' Angry Parenting and child ADHD symptoms in our cross-lagged panel models, however these bi-directional relationships appeared confined to early years (children aged 4- to 7-years) and were more continuous for fathers than for mothers. Although Angry Parenting no longer predicted child ADHD symptoms from wave three, higher child ADHD symptoms continued to predict higher levels of Angry Parenting throughout all waves in a uni-directional manner. Higher child ADHD symptoms at wave 1 (children age 4- and 5-years) also appeared to lead to an overall increase in less-than-optimal parenting (i.e., higher anger, lower warmth and lower consistency) when children were 6- and 7-years and 10- and 11-years of age, however these patterns were not identical. Child ADHD symptoms also predicted the overall increase in Angry Parenting across all waves, as evidenced in LGCM two. Hypothesis 2, that the
bi-directional prospective relationships between less-than-optimal parenting and child ADHD symptoms would be stronger for sons than daughters, was not supported as the modelled relationships did not differ as a function of child gender.

Partial support was also found for Hypothesis 3 that predicted less-than-optimal parenting (i.e., higher scores on Angry Parenting, lower scores on Consistent Parenting, and lower scores on Warm Parenting) at wave one would be predictive of a child ADHD diagnosis at wave three. Only mothers’ Angry Parenting and fathers’ Angry Parenting at wave one were predictive of a child’s ADHD diagnosis at wave three. Hypothesis 4 was not supported as these predictive relationships between parenting and children’s ADHD diagnosis did not differ between sons and daughters.

Mothers and fathers in the current sample appear similar to existing literature in regards to their parenting of sons and daughters. For example, the temporal stability of the autoregressive paths for parenting dimensions in the cross-lagged panel model demonstrate that parenting remains somewhat stable over time (Holden and Miller 1999; Landry et al. 2001). Further, as is commonly found in parenting literature (e.g., Barnett and Scaramella 2015; Russell et al. 1998), non-referred sons and daughters in the current sample received different levels of parenting dimensions, with sons receiving higher levels of less-than-optimal parenting compared to girls (e.g., lower levels of warmth and higher levels of anger).

Prospective Relationships between Parenting and Child ADHD

The results of our cross-lagged panel model, LGCM two, and logistic regression add to the existing evidence regarding the prospective relationships between parenting and child ADHD. These results provide some support for claims (e.g., Johnston and Mash 2001) that a bi-directional relationship exists between child behaviour and parenting. Our results, however, suggest this bi-directional relationship is confined to earlier in childhood and may be more consistent for fathers than mothers, while a uni-directional relationship whereby child ADHD predicts certain parenting dimensions (Angry Parenting in the current sample), continues throughout childhood and into early adolescence. Therefore, our results also support arguments that child behaviour may actually be a stronger or more consistent predictor of parenting across time than the reverse relationship (Barkley 1988; Singh 2003).

As discussed, the most consistent findings that arose from the current analyses relates to the relationship between child ADHD and mothers’ and fathers’ Angry Parenting. Our measure of Angry Parenting related to parental feelings of anger and annoyance towards the child, the level of praise/approval directed towards their child, and the difficulty parents experienced in managing their child’s behaviour. Our findings that Angry Parenting shares a bi-directional relationship with child ADHD symptoms in early years before converting to a uni-directional prospective relationship (child ADHD predicting angry parenting) in middle childhood, is in line with previous suggestions that parenting may become less influential on ADHD at later stages of development (Carlson et al. 1995; Johnston and Mash 2001). The continued influence of child ADHD symptoms on Angry Parenting may have flow-on effects in other domains. For example, parent anger and hostility has been linked to children’s perceptions of parental detachment (Domingos and Bierman 2001), the adoption of a more authoritarian parenting approach (Coplan et al. 2002), parental conflict (Krishnakumar and Buehler 2000) and the development of depression in adolescence (Kaitanen et al. 1999). Further, Angry Parenting behaviours are transmitted generationally, influencing the level of hostility children will later display towards their offspring (Scaramella and Conger 2003). Thus, ADHD treatments that lower ADHD symptoms may assist in also lowering rates of Angry Parenting and the negative outcomes associated with angry parenting within, and across, generations.

Our results extend the findings of previous longitudinal studies regarding parenting and ADHD behaviours; for example, the studies by Keown (2012) and Lifford et al., (2008), by considering (i) bi-directional relationships, and (ii) invariance in these relationships across child genders. However, some differences are notable. Keown found less-than-optimal parenting (i.e., lower positive regard, sensitivity and warmth) was predictive of higher child ADHD symptoms across children 4- to 7-years of age. Within the same age range in the current sample, we found a bi-directional relationship for Angry Parenting, and uni-directional influences of child ADHD symptoms predicting less-positive parenting (i.e., lower Warm and Consistent Parenting). Further, across a 12-month period, Lifford et al. investigated the cross-lagged relationships between parental rejection and ADHD symptoms in 11- to 12-year old children. Lifford et al. found higher levels of paternal rejection predicted higher child ADHD symptoms, while higher rates of child ADHD symptoms predicted higher maternal rejection. This suggests different relationships exist between parenting and child ADHD symptoms depending on whether mothers or fathers are considered. Comparison of the findings of waves four and five of the current study (where children were identical in age to the Lifford et al. study) supports their finding that ADHD symptoms predict fathers’ Angry Parenting, however extends this same finding to mothers.

Several factors may account for the differences between the present results and those of Lifford et al., (2008). First, with both mothers and fathers included in a single, bi-directional model in the current study, as opposed to separate models for mothers and fathers in Lifford et al., less variance was available in each outcome variable for significant prediction in our...
model (i.e., significant relationships were harder to find). However, given the models in the current study were stringently controlled with autoregressive paths and for SES, our findings were sensitive to predictive relationships. Second, our models extended over a longer period (children aged 4- to 13-years), compared to Lifford et al. (children aged from 11- to 13-years). As such, we were able to examine predictive relationships across a broader range of developmental periods. Further, by the ages of 11- to 13-years it is possible the behaviours of the children in Lifford et al. were well established, with parenting having only an incidental, rather than predictive, effect.

The Influence of Child Gender on the Prospective Relationships between Parenting Dimensions and ADHD

Gender differences were found in several aspects of the current results. First, across all waves, boys had significantly higher ADHD symptoms than girls, and were significantly more likely to have a diagnosis of ADHD, consistent with previous findings (Erskine et al. 2013; Willcutt 2012). Gender differences were also found in relation to parenting, with sons more likely to receive higher levels of angry parenting than daughters from both mothers and fathers across all waves. However, no gender differences were found in the predictive relationships examined. Taken together with the finding that sons had higher ADHD symptoms than daughters across all waves, this suggests boys and girls are not parented differently as a function of their gender, but rather, differential parenting may occur as a function of child behaviours. This idea requires further investigation and suggests that future research into the relationship between parenting and gender should consider child behaviour as an influential associate.

No differences between sons and daughters were found in the prospective relationships between parenting and child ADHD in the current study. Thus, although gender differences existed in the mean levels of ADHD symptoms and parenting dimensions, as well as in the rate of diagnosis, it appears ADHD in both boys and girls share the same relationships with mothers’ and fathers’ Angry, Warm and Consistent Parenting. One possibility that may inform future research is that gender differences may arise when comparing boys and girls who receive similar mean levels of parenting and/or have similar levels of ADHD symptoms.

It may be that child gender more closely impacts the relationship between parenting dimensions, other than those investigated here, and child ADHD symptomology (e.g., autonomy support, coercion, control, responsiveness; Holden and Miller 1999; Skinner et al. 2005). Further research is needed. It may also be that child gender impacts the relationship between parenting styles (i.e., the more stable aspects of parenting and the emotional climate in which parenting occurs; Darling and Steinberg 1995) and child ADHD more so than the relationship between parenting dimensions and child ADHD. As parenting dimensions appear to be more influenced by child behaviours than are parenting styles, it may be that parenting styles are more influenced by child gender. For example, the authoritative and/or authoritarian styles may hold significant differences in their relationships to child ADHD based on child gender than do more transient and reactive parenting dimensions.

It has also been suggested that child gender may be a stronger moderator when considering ADHD subtypes rather than ADHD-combined (Baurmeister et al. 2007). Thus, the current study may have failed to find significant child gender differences given our measure of ADHD assessed the combined subtype (i.e., included questions regarding both the inattentive and hyperactive/impulsive). It may be fruitful for future research to examine ADHD subtypes when exploring child gender differences in prospective relationships between parenting and child ADHD.

Limitations

The authors acknowledge limitations of the current study. It is possible the mother-report measurement of child ADHD symptomology on the SDQ may have overestimated/underestimated symptoms. Despite the established validity and reliability of the SDQ, previous research has demonstrated some inconsistency between SDQ ratings between informants (e.g., mother, father, teacher, self; Stokes et al. 2014). Thus, different relationships may have been found in our models had different ADHD informants been used. Further, ratings of parenting dimensions and child ADHD symptoms relied on self-report and informant-report, respectively. This may have impacted the validity and reliability of ratings compared to a more objective measure of these variables, such as observational assessment. Identification of the clinical sample was also based on parent report, and although a further question regarding medication use was used in an attempt to verify clinical status, it is not possible to determine reliability of self-reported diagnosis.

There is also Type I error potential given the multiple analyses and pathways in the current study. However, confidence that true relationships were found is increased by (i) the relationships being in the expected directions, and (ii) the fact significant results were found despite the comprehensive autoregressive/control model. In order to examine both maternal and paternal parenting styles, non-nuclear families (e.g., single-parent families, same-sex parents) were excluded from the current analysis, thus precluding generalisation of our findings to non-nuclear families. Unfortunately, limitations
Exploring gender differences in ED prevalence

Acknowledgements

References


Landy, S. H., Smith, K. E., Swain, P. R., Azen, M. A., & Valler, S. (2001). Does early responsive parenting have a special importance for children’s development or is consistency across time early childhood necessary? Developmental Psychology, 37, 387-403.


Exploring gender differences in ED prevalence


## Appendix C

### List of full text studies excluded for study 2

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Reason for Exclusion</th>
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<td>Adewuya &amp; Famuyiwa (2007)</td>
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<td>Almqvist, et al. (1999)</td>
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<td>Angold &amp; Costello (1996)</td>
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<td>Buffers, Dougherty, Carlson, Rose &amp; Klein (2012)</td>
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## Appendix D

### Model estimate results for cross-lagged panel analysis

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### Exploring gender differences in ED prevalence

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Exploring gender differences in ED prevalence

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Coding: W1 = Wave 1 (children aged 4-5); W2 = Wave 2 (children aged 6-7); W3 = Wave 3 (children aged 8-9); W4 = Wave 4 (children aged 10-11); W5 = Wave 5 (children aged 12-13); MWARM = Mother warm parenting; FWARM = Father warm parenting; MCON = Mother consistent parenting; FCON = Father consistent parenting; MANG = Mother angry parenting; FANG = Father angry parenting; SES = Socio-economic status.
Appendix E

Model estimate results for latent growth curve model 1 - parenting practices as outcomes

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Coding: (i)=intercept; (s)=slope
Exploring gender differences in ED prevalence

**Appendix F**

**Model estimate results for latent growth curve model 2 – ADHD symptoms as outcome**

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</tr>
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Coding: (i)=intercept; (s)=slope


**Appendix G**

**Results for logistic regression**

<table>
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<tr>
<th>Predictor</th>
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<th>SE B</th>
<th>e^b</th>
<th>p-value</th>
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<td>0.09</td>
<td>1.01</td>
<td>0.91</td>
</tr>
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<td>Father Consistent Parenting</td>
<td>0.03</td>
<td>0.08</td>
<td>1.03</td>
<td>0.70</td>
</tr>
</tbody>
</table>

Coding: e^b = exponentiated B
INVITATION TO PARTICIPATE IN A DEAKIN UNIVERSITY RESEARCH PROJECT

Dear [name]

Deakin University is currently conducting a research project investigating the diagnostic decision making processes of clinicians when they are assessing children for psychological disorders. As part of this project, we are seeking participants who may assess, diagnose, or treat psychological disorders in children as part of their profession, including [psychologists, psychiatrists, general practitioners, paediatricians]. Therefore, we would like to invite members of [organisation] to participate in this research.

Participation in this project will involve the completion of two brief online surveys, completed approximately two weeks apart. Each survey takes approximately 5-10 minutes to complete. Survey one will collect basic demographic information (e.g., age, sex, years practicing). Participants will then be presented with a case vignette of a child which describes a certain set of behavioural and emotional issues. Participants will be asked for their opinion on what disorder the case represents. Approximately two weeks after completing the first survey participants will be emailed directly by the research team with a weblink to the second survey.

Participants will be required to provide their email address when completing the first survey. This email address will be used solely for the purposes of providing participants with the link to the second survey. Email addresses will not be added to any Deakin databases or mail-out lists, nor will they be passed on to any third parties. All email addresses will be deleted from participant records once the second survey has been completed, making all data non-identifiable.
The Organisation’s responsibilities: The responsibilities your organisation holds in participation in this project are minimal and will hopefully cause minimal disruption. We seek consent to contact members of [organisation] via email or an online mailing/listing prepared by the research team but distributed by your organisation. This may be in the form of an individual email regarding the project that is sent to members, or alternatively, may be included in any regular email sent to members (e.g., a monthly newsletter). The research team is happy to discuss any possible variations on providing information regarding this project to members of your organisation. The email and/or website listing would provide participants with a weblink directly to the online survey. Your organisation’s and member’s participation in this study is voluntary. Member’s are free to withdraw their participation up until the time they have completed the second survey, and your organisation is free to withdraw its participation up until the time the data are processed.

Attached to this letter are the following:

1. An Organisational Plain Language Statement
2. An Organisational Consent Form
3. An Organisational Withdraw of Consent Form

Please read the Organisational Plain Language Statement carefully before making a decision regarding participation in this project. Following this, if your organisation is willing to participate and to make the weblink for this project available to its members, please sign the Organisational Consent Form and either email it to [email address] or fax or post it to the details listed at the bottom of this letter. If you have any questions regarding participation, or the project in general, please do not hesitate to contact a member of the research team via the details below.

Kind Regards,
Appendix I

Study 4 plain language statement and consent forms for organisations

PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: {insert name of organisation}

Organisational Plain Language Statement

Date:

Full Project Title: The Diagnostic Decision Making Processes of Clinicians when Assessing Children for Psychological Disorders

Principal Researcher: Dr. Merrilyn Hooley, PhD

Student Researcher: Mr. David H. Demmer BPsych(Hons) DPsych (Clinical) candidate

Recent research (e.g., Bruchmuller, Margraf & Schneider, 2012) has suggested that clinicians use heuristics (i.e., cognitive shortcuts), rather than DSM-based criteria when assessing and children for psychological disorders. This use of heuristics may impact on the appropriateness of diagnoses, and result in over- and under-diagnoses of some disorders. We seek to explore the heuristics that
exploring gender differences in ED prevalence

clinicians use in their assessment of psychological disorders in children via an online survey

We invite members of the [insert name of organisation] to take part in this research project. In order to take part participants must be [insert required qualifications for profession].

This Plain Language Statement (PLS) contains information about the project. Its purpose is to explain as openly and clearly as possible all the procedures involved so that you can make a fully informed decision about your organisation’s participation.

If you agree to take part in this study, you will be asked to sign the attached Consent Form and return to the address below. By signing the Consent Form, you indicate that you understand the information, and that you give your consent to provide information about this project to members of your organisation.

Information regarding this project:

1. Purpose: The aim of this project is to investigate diagnostic criteria in the diagnosis of psychological disorders in childhood.

2. Methods: The methods used for this investigation will be two brief online-only surveys. Each survey should take approximately 5-10 minutes to complete. Approximately two weeks after completing the first survey participants will be sent a direct email with a link to the second survey.

3. Participants will be required to supply their email address when completing the first survey. This will be used solely for the purposes of emailing participants the link to the second survey. Participants email addresses will not be added to any Deakin databases or mail-out lists, nor will it be passed on to any third parties. All email addresses will be deleted from participant records once the second survey has been completed to ensure the confidentiality of the data supplied.

4. There are no expected risks, potential benefits or adverse effects to participants.
5. Participant privacy and confidentiality will be protected by the de-identification of all data; no individual data will be reported.

6. Member’s participation in this study is voluntary. Members are free to withdraw their participation up until the time they have completed the second survey.

1. The results of this investigation will be submitted for publication to a peer-reviewed journal. Preliminary results will also be available to participants via a weblink, and to your organization should you so indicate on the attached consent form.

Should you require any further information, have any queries or problems, or wish to withdraw your participation, you can contact on the Principle Investigator via the following details:

Dr. Merrilyn Hooley PhD
School of Psychology
Deakin University
221 Burwood Highway,
Burwood, 3125
Ph: B/H 03 9244 6499
Email: merrilyn.hooley@deakin.edu.au

Complaints

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact:

The Manager, Ethics and Biosafety, Deakin University, 221 Burwood Highway,
Burwood Victoria 3125, Telephone: 9251 7129, research-ethics@deakin.edu.au

Please quote project number 201_2014.
PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: [insert organisation here]

Organisational Consent Form

(To be used by Organisational Heads providing consent for

staff/members/patrons

to be involved in research)

Date:

Full Project Title: The Diagnostic Decision Making Processes of Clinicians

when Assessing Children for Psychological Disorders

Reference Number: 201_2014

I have read and I understand the attached Plain Language Statement.

I give my permission for members of [insert organisation here] to participate in this project according to the conditions in the Plain Language Statement, and to support the recruitment of members for participation in this project.

I have been given a copy of Plain Language Statement and Consent Form to keep.

The researcher has agreed not to reveal the participants’ identities and personal details if information about this project is published or presented in any public form.

I agree that
1. The [insert organisation here] MAY / MAY NOT (please circle one) be named in research publications or other publicity without prior agreement.

2. The [insert organisation here] EXPECTS/ DOES NOT EXPECT (please circle one) to receive a copy of the research findings or publications.

Name of person giving consent (printed)

..............................................................

Signature .....................................................

Date  .....................................................
PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: [insert organisation here]

Organisation Withdrawal of Consent Form

(To be used for Organisations who wish to withdraw from the project)

Date: 

Full Project Title: The Diagnostic Decision Making Processes of Clinicians when Assessing Children for Psychological Disorders

Reference Number: 201_2014

I hereby wish to WITHDRAW my consent to participate in the above research project and understand that such withdrawal WILL NOT jeopardise my relationship with Deakin University.

Participant’s Name (printed) ............................................................

Signature ....................................................................................... 

Date .........................
Please mail or fax this form to:

Dr. Merrilyn Hooley PhD
School of Psychology
Deakin University
221 Burwood Highway,
Burwood, 3125
Ph: B/H 03 9244 6499
Fax: 03 9244 6858
Email: merrilyn.hooley@deakin.edu.au
Appendix J

Study 4 plain language statement for participants

PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: Clinicians

<table>
<thead>
<tr>
<th>Plain Language Statement</th>
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<tbody>
<tr>
<td><strong>Date:</strong> 02/07/2015</td>
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<tr>
<td><strong>Full Project Title:</strong> The Diagnostic Decision Making Processes of Clinicians when Assessing Children for Psychological Disorders</td>
</tr>
<tr>
<td><strong>Principal Researcher:</strong> Dr. Merrilyn Hooley, PhD</td>
</tr>
<tr>
<td><strong>Student Researcher:</strong> Mr. David H. Demmer BPsys(Hons) DPysch (Clinical) candidate</td>
</tr>
</tbody>
</table>

You are invited to take part in this research project exploring the diagnostic decision-making processes of clinicians when assessing children for psychological disorders. In order to participate, you must be one of the following: (i) a Psychologist registered with AHPRA (provisionally or fully), (ii) a Paediatrician registered with the RACP (Basic or Advanced Trainee, or Fellow), (iii) a Psychiatrist registered with RANZCP (Trainee or Fully), or (iv) a General Practitioner registered with RACGP (Registrar, Resident, Intern, or Full- or Part-time GP). You may meet one or more of these categories.

This Plain Language Statement (PLS) contains information about the project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project so that you can make a fully informed decision whether you are going to participate.

Please read this PLS carefully. Once you understand what the project is about, and if you agree to take part in it, please click the 'I accept' button at
Exploring gender differences in ED prevalence

the bottom of this page to begin. Please note that by clicking the ‘I accept’ button you are consenting to participate in this project.

**Information regarding this project:**

- **Purpose:** The aim of this project is to investigate diagnostic criteria used in the diagnosis of psychological disorders in children.
- **Methods:** The methods used for this investigation will be two brief online-only surveys. Each survey should take approximately 5-10 minutes to complete. You will be taken to the first survey by clicking the ‘I accept’ button below. Approximately two weeks after completing the first survey you will be sent an email with a link to the second survey.
- **You will be required to supply your email address when completing the first survey. This will be used *solely* for the purposes of emailing to you the link to the second survey. Your email address *will not* be added to any Deakin databases or mail-out lists, nor will it be passed on to any third parties. Your email address will be deleted from your participant record once the second survey has been completed to ensure the confidentiality of the data you supplied.
- **There are no expected risks, potential benefits or adverse effects to participants.**
- **Expected benefits to the professional community involve a better understanding of the diagnostic criteria used in the diagnosis of common childhood psychological disorders.**
- **Your privacy and confidentiality will be protected by the de-identification of all data; no individual data will be reported.**
- **Your participation is voluntary and you can withdraw at any point up until you have completed the second survey.**
- **The results of this investigation will be submitted for publication to a peer-reviewed journal. Preliminary results will also be available via the following web link by February 2016:**


If you require any further information, have any queries or problems, or wish to withdraw your participation, you can contact on the Principle Investigator via the following details:

Dr. Merrilyn Hooley PhD

School of Psychology
Complaints
If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact:

The Manager, Ethics and Biosafety, Deakin University, 221 Burwood Highway, Burwood Victoria 3125, Telephone: 9251 7129, research-ethics@deakin.edu.au

Please quote project number [201_2014].
Appendix K

Study 4 online survey time 1

Q. Please enter your email address. Your email address is needed in order to send you part two of this survey in approximately two weeks.

Q. Which one of the following professions are you? (If you meet several of these, please select the one you have been for the longest)

- Psychologist provisionally or fully registered with AHPRA
- Paediatrician registered with The Royal Australasian College of Physicians as a Basic or advanced trainee, or a Fellow
- General Practitioner registered with The Royal Australian College of General Practitioners as either a Registrar, Resident, Intern, or Full- or Part-time GP
- Psychiatrist registered (Trainee or Fully) with the Royal Australian and New Zealand College of Psychiatrists

How did you receive the link to this survey?

- From my professional association
- From a colleague

Gender

- Male
- Female

Age (in years):

How competent do you feel working with child and adolescent clients?

1 2 3 4 5 6 7 8 9 10
Not competent Very competent

What is your current child and adolescent caseload?

- 0 – 25%
- 26 – 50%
- 51 – 75%
- 75 – 100%
<Questions for psychologists>

What is your current AHPRA registration?

- Provisionally registered
- Fully registered

What is your highest level of education in psychology?

- Currently completing 4+2 Model (completed undergraduate degree, honours in psychology, and am currently completing 2 years of supervised practice)
- Completed 4+2 Model (completed undergraduate degree, honours in psychology, and have completed 2 years of supervised practice)
- Currently completing Master’s Degree in psychology
- Completed Master’s Degree in psychology
- Currently completing Doctoral Degree in Psychology
- Completed Doctoral Degree in Psychology
- Currently completing combined Masters/PhD in Psychology
- Completed combined Masters/PhD in Psychology
- Other – please specify:

Are you an endorsed member of any of the following APS colleges?

- Clinical Neuropsychologists
- Clinical Psychologists
- Community Psychologists
- Counselling Psychologists
- Educational and Developmental Psychologists
- Forensic Psychologists
- Health Psychologists
- Organisational Psychologists
- Sport and Exercise Psychologists
- I do not belong to any of these specializations/colleges

How many years have you spent as a practising psychologist?:

<Questions for paediatricians>

What is your current RACP registration?

- Basic trainee
- Advanced trainee
- Fellow

How many years have you spent as a practising paediatrician?:
<Questions for GPs>
What is your current RACGP registration?
- Registrar
- Resident
- Intern
- Full-time GP
- Part-time GP

How many years have you spent as a practising GP?:

<Questions for psychiatrists>
What is your current RANZCP registration?
- Trainee
- Fully

How many years have you spent as a practising Psychiatrist?:

Please carefully read the following case vignette and then answer all of the questions below.

<INSERT CASE VIGNETTE HERE>

Do you believe this child meets diagnosis for a DSM 5 psychological disorder?
- Yes
- No

If yes, please state the DSM 5 diagnosis that you believe the child meets (Provide only ONE diagnosis):

If no, what would your working hypothesis be for further exploration (i.e., what disorder do you think the child is most likely to have based on the vignette – provide only ONE diagnosis):

What were the most salient/important features of this child’s presentation on which you based either your diagnosis or your working hypothesis?:
Aside from your diagnosis or your working hypothesis, do you believe there are any comorbid DSM 5 disorders, or features of any other DSM 5 disorders present? If so, please list these here – you can list as many disorders as you feel appropriate:

How confident are you that your diagnosis or working hypothesis is correct?

1 2 3 4 5 6 7 8 9 10
Not confident Very confident

Thank you for your participation in part one of this project. In approx. two weeks the research team will email you a weblink to complete part two of this survey.

We encourage participants to forward the link for this survey to their colleagues to participate.
Appendix L

Online survey time 2

Q. Please re-enter your email address in order to link your responses from this survey to your responses in survey one:

<INSERT CASE VIGNETTE HERE>

Q. Do you believe this child meets diagnosis for a DSM 5 psychological disorder?
   o Yes
   o No

Q. If yes, please state the DSM 5 diagnosis that you believe the child meets (Provide only ONE diagnosis):

Q. If no, what would your working hypothesis be for further exploration (i.e., what disorder do you think the child is most likely to have based on the vignette – provide only ONE diagnosis):

Q. What were the most salient/important features of this child’s presentation on which you based either your diagnosis or your working hypothesis?:

Q. Aside from your diagnosis or your working hypothesis, do you believe there are any comorbid DSM 5 disorders, or features of any other DSM 5 disorders present? If so, please list these here – you can list as many disorders as you feel appropriate:

Q. How confident are you that your diagnosis or working hypothesis is correct?
   1  2  3  4  5  6  7  8  9  10
   Not confident
   Very confident

You have now completed your participation in this project, thank you for your involvement.
We encourage participants to forward the link for this survey to their colleagues to participate. Please forward this link via email.
Appendix M
Study 4 vignettes

Vignette 1. Female. ADHD-C diagnosis fulfilled, Major Depressive Disorder missing 2 symptoms.

Sarah is a ten-year-old girl who currently attends her local primary school. Sarah’s parents state that Sarah has had a lot of energy ever since she was a toddler. Sarah remarks that she has trouble sitting still because it always makes her feel uncomfortable. Sarah has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Sarah’s parents and teachers has noticed her mood has become ‘consistently sad and low’ over the last eight months, with Sarah often appearing on the verge of tears. As a consequence, she has been avoiding tasks such as homework, therefore she is falling behind in her schoolwork, and is at risk of being held back a grade.

Sarah repeatedly gets in trouble in class for yelling out answers to questions before the teacher has finished talking, and for constantly leaving her seat. Sarah’s teacher reports that she is very physically agitated and fidgety, and easily distracted by sounds and activities around her. Sarah has difficulties with her peers because she struggles to understand boundaries; for example, she is constantly butting into games that other children are playing during lunch time and finds it difficult to wait until it is her turn to play. As a consequence, Sarah finds it difficult to make, and sustain, friendships.

Sarah has become very forgetful (e.g., forgetting to pack her school bag with all the items needed for her day at school, forgetting to make her bed), and often loses items (e.g., school books, toys, and her toothbrush), and her parents are frustrated at continually having to replace these items. Sarah’s parents and her teachers also note that she rarely listens when being spoken to. Sarah’s care team (including a psychologist, paediatrician and GP) have ruled out any general medical or psychotic conditions.
Vignette 2. Female. Borderline. ADHD-C missing 1 hyperactive/impulsive symptoms and 1 inattentive symptoms, Major Depressive Disorder missing 1 symptom.

Sarah is a ten-year-old girl who currently attends her local primary school. Sarah’s parents state that Sarah has had a lot of energy ever since she was a toddler. Sarah remarks that she has trouble sitting still because it always makes her feel uncomfortable. Sarah has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Sarah’s parents and teachers have noticed her mood has become ‘consistently sad and low’ over the last eight months, with Sarah often appearing on the verge of tears. As a consequence, she has been avoiding tasks such as homework, therefore she is falling behind in her schoolwork and is at risk of being held back a grade.

Sarah repeatedly gets in trouble in class for leaving her seat. Sarah’s teacher reports that she is very physically agitated and fidgety, and easily distracted by sounds and activities around her. Sarah has difficulties with her peers because she struggles to understand boundaries; for example, she is constantly butting into games that other children are playing during lunch time and finds it difficult to wait until it is her turn to play. As a consequence, Sarah finds it difficult to make, and sustain, friendships.

Sarah has become very forgetful (e.g., forgetting to pack her school bag with all the items needed for her day at school, forgetting to make her bed), and often loses items (e.g., school books, toys, and her toothbrush), and her parents are frustrated at continually having to replace these items. During the past eight months Sarah has been having trouble getting to sleep and staying asleep. Sarah’s care team (including a psychologist, paediatrician and GP) have ruled out any general medical or psychotic conditions.
Vignette 3. Female. ADHD-C missing 2 hyperactive/impulsive symptoms and 2 inattentive symptoms, Major Depressive Disorder diagnosis fulfilled.

Sarah is a ten-year-old girl who currently attends her local primary school. Sarah’s parents state that Sarah has had a lot of energy ever since she was a toddler. Sarah remarks that she has trouble sitting still because it always makes her feel uncomfortable. Sarah has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Sarah’s parents and teachers have noticed her mood has become ‘consistently sad and low’ over the last eight months, with Sarah often appearing on the verge of tears.

Sarah’s teacher reports that she is very physically agitated and fidgety, and easily distracted by sounds and activities around her. Sarah has difficulties with her peers because she struggles to understand boundaries; for example, she is constantly butting into games that other children are playing during lunch time and finds it difficult to wait until it is her turn to play. As a consequence, Sarah finds it difficult to make, and sustain, friendships.

Sarah has become very forgetful (e.g., forgetting to pack her school bag with all the items needed for her day at school, forgetting to make her bed), and often loses items (e.g., school books, toys, and her toothbrush), and her parents are frustrated at continually having to replace these items. During the past eight months Sarah has been having trouble getting to sleep and staying asleep. Sarah’s care team (including a psychologist, paediatrician and GP) have ruled out any general medical or psychotic conditions; however they are concerned that she has not reached an appropriate weight for a girl her age.
Vignette 1. Male. ADHD-C diagnosis fulfilled, Major Depressive Disorder missing 2 symptoms.

Simon is a ten-year-old boy who currently attends his local primary school. Simon’s parents state that Simon has had a lot of energy ever since he was a toddler. Simon remarks that he has trouble sitting still because it always makes him feel uncomfortable. Simon has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Simon’s parents and teachers have noticed his mood has become ‘consistently sad and low’ over the last eight months, with Simon often appearing on the verge of tears. As a consequence, he has been avoiding tasks such as homework, therefore he is falling behind in his schoolwork, and is at risk of being held back a grade.

Simon repeatedly gets in trouble in class for yelling out answers to questions before the teacher has finished talking, and for constantly leaving his seat. Simon’s teacher reports that he is very physically agitated and fidgety, and easily distracted by sounds and activities around him. Simon has difficulties with his peers because he struggles to understand boundaries; for example, he is constantly butting into games that other children are playing during lunch time and finds it difficult to wait until it is his turn to play. As a consequence, Simon finds it difficult to make, and sustain, friendships.

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Vignette 2. Male. Borderline. ADHD-C missing 1 hyperactive/impulsive symptoms and 1 inattentive symptoms, Major Depressive Disorder missing 1 symptom.

Simon is a ten-year-old boy who currently attends his local primary school. Simon’s parents state that Simon has had a lot of energy ever since he was a toddler. Simon remarks that he has trouble sitting still because it always makes him feel uncomfortable. Simon has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Simon’s parents and teachers have noticed his mood has become ‘consistently sad and low’ over the last eight months, with Simon often appearing on the verge of tears. As a consequence, he has been avoiding tasks such as homework, therefore he is falling behind in his schoolwork and is at risk of being held back a grade.

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Vignette 3. Male. ADHD-C missing 2 hyperactive/impulsive symptoms and 2 inattentive symptoms, Major Depressive Disorder diagnosis fulfilled.

Simon is a ten-year-old boy who currently attends his local primary school. Simon’s parents state that Simon has had a lot of energy ever since he was a toddler. Simon remarks that he has trouble sitting still because it always makes him feel uncomfortable. Simon has always shown difficulty sustaining attention on most tasks for an extended period of time, however lately is finding it difficult to concentrate nearly every day. Further, Simon’s parents and teachers have noticed his mood has become ‘consistently sad and low’ over the last eight months, with Simon often appearing on the verge of tears.

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